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PRACTICAL CONSIDERATIONS IN THE DIAGNOSIS OF PERIPHERAL NERVE INJURIES, WITH SPECIAL REFERENCE TO COMPENSATORY MOVEMENTS

SAMUEL D. INGHAM, M.D., AND JOHN H. ARNETT, M.D.

PHILADELPHIA

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I. INTRODUCTION

During the year of its active existence at Cape May, there passed through the U. S. Army General Hospital No. 11, at which the authors were stationed, 617 cases of peripheral nerve wounds. When the entire neurosurgical staff was transferred to Fox Hills, Staten Island, N. Y. (U. S. Army General Hospital No. 41), a large number of additional peripheral nerve cases were observed. These cases were studied with considerable care, and the findings fully recorded on forms especially prepared by the members of the Cape May neurosurgical staff. These forms were later adopted throughout the army for use in peripheral nerve hospitals. Shortly after admission each patient was examined by his ward surgeon and discussed at a conference of the neurosurgical staff. Re-examinations were made and the patients reviewed at conferences at intervals of from one to two months. In many instances patients have been operated on and the clinical diagnosis studied at this time in the light of the operative findings. By this procedure errors in diagnosis have been reduced to a minimum. The average case has remained under observation over three months so that ample opportunity for study has been afforded. The patients have all reached us some time after the occurrence of the wound, however, so that the observations which follow must not be taken to apply to early nerve wounds, but to injuries of over a month's standing.

As a result of these studies, some points have been noted which have not been sufficiently emphasized, or have escaped mention in descriptions of typical paralyses from nerve wounds. This paper will deal with this practical aspect of the subject rather than attempt a systematic discussion of nerve injuries, or the setting forth of debated theories.

II. FACTORS CONCERNED IN THE DIAGNOSIS OF PERIPHERAL NERVE LESIONS

A complete diagnosis should include the following points:

1. Identification of the nerve injured.
2. Description of the level at which the lesion occurs.
3. A statement of the degree of functional loss in the injured nerve.
4. Some note as to the status of recovery.
5. An estimate of the pathologic condition.

A. Slight Motor Function Distinguished from Complete Paralysis.—The first and second parts of the diagnosis generally present little difficulty. The fourth point is merely a matter of comparing the degree of function found at different times. The fifth (to determine the pathology existing in the nerve) is frequently impossible before operation. We are, therefore, particularly concerned with the third part of the diagnosis, for on this depends the whole question of

treatment. It is nearly always possible to distinguish between complete loss of function, and the first faint traces of functional return. For correct observation on this point the patient looks to his surgeon to shield him from months of delay on the one hand, and unnecessary operation on the other.

B. Relative Value of Various Observations.—Although we have studied this problem from almost every angle, in the hope of finding some sure guide in the diagnosis of nerve injuries, experience has taught us to depend largely on the simpler diagnostic signs as being the most reliable. In the order of importance these may be rated: (1) voluntary movement; (2) sensation; (3) electrical reactions, and (4) trophic phenomena.

Fig. 1.—The typical hand of ulnar paralysis. The interosii are wasted, and the little and ring fingers flexed.

1. *Trophic Phenomena.*—First, it has been interesting to note that the trophic disturbances—the least important diagnostic sign—often enable one to make the diagnosis the instant the patient enters the room. So characteristic is the hypothenar and interosseous muscle atrophy in the ulnar cases, and the thenar atrophy in median injuries, that by examining plaster casts of the hands, one can pick out the median or ulnar cases nine times out of ten, provided brachial plexus lesions are excluded (Fig. 1). The trophic condition of the hand is, however, of very little value in showing the early signs of returning function. Indeed there may be the most pronounced degree of atrophy of the interosii, and yet all movements be executed with the intrinsic hand muscles apparently as well as though the hand were normal (Fig. 2).

2. *Electrical Reactions.*—Electrical examination has proved a disappointment to surgeons as it has failed to determine conclusively the degree of nerve injury or of nerve recovery. In two ways however, it is of real value. The faradic current generally settles quickly and definitely the differential diagnosis between a functional and an organic nerve lesion. If there is any question of the existence of a functional basis for a paralysis, and faradic stimulation gives a response in the supposedly paralyzed muscles, it is most unlikely that the paralysis is due to organic changes. Another important use of the electric current is in determining whether a muscle which now contracts, was once paralyzed. It has been observed that a recovering muscle often gives a slow, wavy response to galvanic stimulation, with

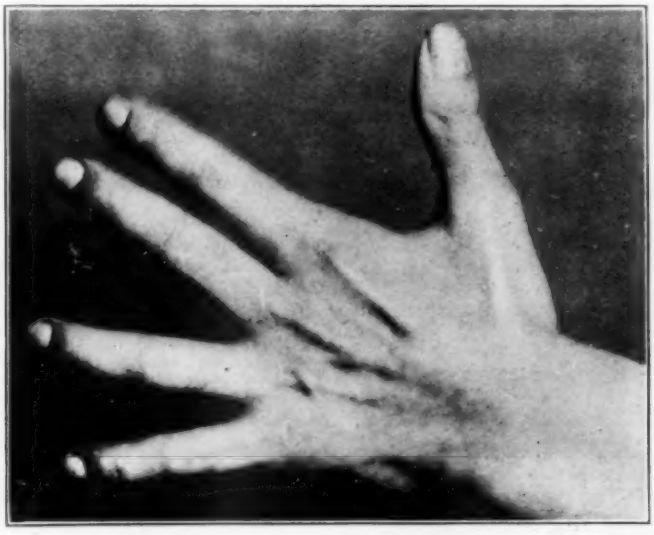

Fig. 2.—Ulnar atrophy in a case of ulnar paralysis in which function was returned in all ulnar muscles.

a tendency toward a reversal of formula, even after it contracts voluntarily and responds to the faradic current. Therefore such a response as this, in a muscle which exhibits voluntary contraction, affords evidence that there was once paralysis. Attempts to determine the degree or nature of the injury by means of the electric current have, however, resulted in disappointment, since the familiar reactions of degeneration are frequently present in connection with spontaneous recovery, and may not be exhibited in their entirety in cases where the nerve has been completely severed.

3. *Sensory Changes.*—The sensory examination is of considerable value, but unless each examination is made by the same observer, or with standardized instruments, the margin of error is apt to be wide.

In routine examinations for impairment of sensation, all but three sensory tests have been discarded. Pressure, pain and tactile sensation are tested, the esthesiometers devised by one of the authors (S. D. I.) being found the most satisfactory (Fig. 3). These esthesiometers were adopted as part of the neurologic equipment of the army peripheral nerve hospitals. Standard charts are used, and loss of deep pressure sense is shown by solid black. Tests for this loss are made by an esthesiometer which exerts a pressure of 1,000 gm. over an area of 0.25 sq. cm. Loss of pain sense is recorded by means of stippling within the anesthetic area which has been outlined on the chart. For this test an esthesiometer is used by means of which a pressure of 15 gm. is exerted on a sharp needle. Superficial tactile sensation is tested by means of a soft artist's brush, so trimmed that pressure over the most delicate portion of the skin does not cause visible depression of the epidermis. Loss of superficial tactile sensa-

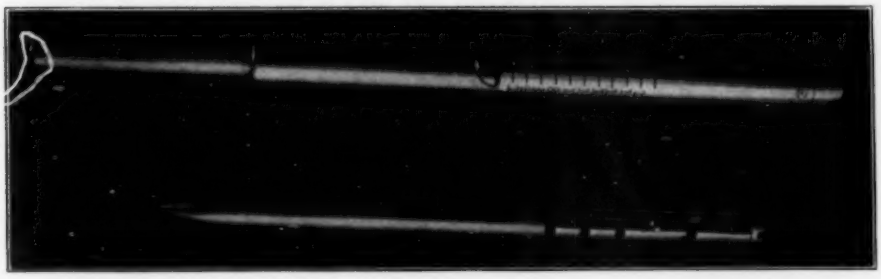

Fig. 3.—Esthesiometers devised by one of the authors, and used in the army peripheral nerve hospitals. The instrument above tests deep sensation, that below tests superficial pain sensation.

tion is charted by means of hatching within the outline of the anesthetic area. Even when thus reduced to their simplest terms, sensory examinations require more time than all the other examinations combined; moreover, the sensory is of less value than the motor examination except in paralyses of purely sensory nerves, and in certain cases where returning sensation is the first evidence of recovery or regeneration.

The test described by Tinel, consisting of distal tingling on percussion over a regenerating nerve trunk, has been found to be of confirmatory value.

Another phase of the sensory examination deserves mention, namely, spontaneous pain. When this pain is severe and burning in character and is increased by touching the skin over the painful area, it is known as causalgia. This symptom is considered as evidence of an irritative lesion involving the sympathetic nerves. The presence

of causalgia, however, by no means precludes the possibility of an anatomic division of the nerve. In two of our cases causalgia was observed shortly after suture of the entire sciatic nerve.

4. *Motor Loss.*—Most important is the examination of motor function, just as motor paralysis constitutes the greatest part of the patient's disability from the standpoint of utility. Three observations are possible in testing the motor function of muscles: the effect of muscular contraction on the tendon, the effect on the joint and the effect on the muscle itself.

(*a*). Tendon Palpation.—Observing the effect of muscle contraction on the tendon by palpation is the most accurate method when it can be utilized. The observer may, however, be misled by the tightening of the tendon due to a change in the position of the joint, the resulting passive tension on the tendon being mistaken for the effect of muscle action. To eliminate such errors it is well so to fix the joint that no movement can occur, and then to have the patient attempt to move the joint. A tendon can in this way be made to stand out so as to be readily perceived, provided its muscle contracts. The factor of passive pull has been eliminated because the joint is immobilized.

(*b*). Observations of the Muscle.—We may dismiss with a few words the subject of observation on muscles. It is generally most unsatisfactory to attempt to see or feel the contraction over the belly of a muscle. Muscles in the vicinity, if they contract, will give an erroneous impression of action in the paralyzed muscle, while a slight true contraction will pass unnoticed. Exceptions to this rule are found in the muscles of the thenar and hypothenar groups, the supinator longus and the deltoid. Palpation and inspection of these muscles furnish very valuable evidence of muscle function.

(*c*) *Joint Action.*—The observation of joint movements is of great value in testing the function of muscles, but the interpretation of this observation is apt to be difficult, and erroneous conclusions may easily be drawn. For example, we might conclude that a patient necessarily had musculospiral function because he could extend the wrist. This is by no means the case. Probably all who have had an opportunity to watch patients with musculospiral lesions have seen many who obtain a certain degree of wrist extension, the musculospiral nerve being completely severed (Fig. 4). Extension is here produced by the action of muscles ordinarily not concerned in this movement. Actions of this type are generally designated as compensatory movements, and are particularly prone to occur in patients who have had paralysis for a long time, patients who have received special training in the use of their muscles, or patients in whom muscle contractures have occurred. Compensatory movements may be due either to the

contraction of one set of muscles or to the relaxation of another. They may also be due either to the factors of gravity or momentum.

III. PRODUCTION OF COMPENSATORY MOVEMENTS

A. *Compensatory Movements Produced by Momentum.* — To understand the part which momentum plays in the production of compensatory movements, one may observe the patient with a fracture of the patella and separation of the fragments. He obtains extension of the knee by a swinging movement, thus substituting momentum of the leg for contraction of the quadriceps extensor muscle. The patient with musculospiral paralysis uses momentum in a similar manner, and frequently acquires considerable facility in extending the wrist by giving the hand a quick backward flip. For this reason joint action should not be taken as evidence of muscle contraction unless the movement is carefully analyzed by the observer, and the part so immobilized as to eliminate the momentum factor.

Fig. 4.—A case of complete musculospiral paralysis extending the wrist. This is accomplished by winding up the tendons of the extensor muscles (which have undergone contracture) by flexing the fingers.

B. *Gravity as a Factor.*—Gravity must also be considered as a means of producing compensatory movements. In musculospiral paralysis, the limp wrist which cannot be extended against gravity, falls into the position of extension if the hand is turned in supination. In order that the test may be perfectly reliable in showing all the motion that the patient is able to make, and at the same time rule out confusing movements due to gravity, the hand should be held midway between pronation and supination — the zero position. What is true of the wrist is also true of other joints; tests for muscle action should always be made with the joint in the zero position.

C. *Relaxation of Functioning Muscles.*—Far more confusing, however, than the movements produced by momentum or gravity, are those produced by other muscles which simulate the action of the paralyzed ones. Let us assume that we have a case of complete musculospiral paralysis with shortening in the extensor muscles, so that the hand naturally assumes the position of extension instead of wrist drop. If one were to grasp the hand and cause the wrist to flex, the hand would spring back into its extended position as soon as it was released. The patient can obtain the same result by the action of the flexors. These muscles pull the hand down, and when relaxation occurs, the hand again assumes the attitude of extension. When this movement is seen for the first time it gives a very misleading impression of extensor action, whereas the whole movement is really due to the flexors. Since the extension of the wrist is due to relaxation of the flexors instead of contraction of the extensors, it may be designated as a compensatory movement of relaxation. To eliminate this possible source of error, the flexors should be relaxed before the movement of extension is attempted. This is best accomplished by having the patient flex the wrist, slowly relax and then attempt slow, steady extension. No further movement of relaxation can occur under these conditions because the flexors are already completely relaxed. Any further movement of extension would therefore be evidence of musculospiral action, provided the relative position of the fingers and hand remains unchanged. The reason for this provision will be considered in the next paragraph.

D. *Contraction of Nonparalyzed Muscles.*—Let us again assume that we have a complete musculospiral paralysis, with contractures in the extensors of sufficient degree to prevent the hand from falling into the wrist drop position. If the examiner were to grasp the fingers and flex all of the phalanges until the hand assumed the position of a tightly closed fist, the extensor tendons, running over the summits of the knuckles, would be made more tense and would prevent the fingers from flexing unless some change in joint position could be secured which would lessen the tension. The wrist joint is the only one free to move which could release this tension. Extending the wrist joint tends to relax the extensors. This movement must therefore occur to allow the flexion of the fingers when the extensors have undergone contracture; and, indeed, this movement does occur when a patient with such contractures attempts to close the fist tightly, so that we have the curious spectacle of a man with musculospiral paralysis extending the wrist by flexing the fingers (Fig. 4). This type of movement may be characterized as a compensatory movement of contraction. On account of this movement of substitution, while testing

for wrist extension as an evidence of musculospiral action, the relative position of the fingers and the hand must remain unchanged.

E. *Elimination of Compensatory Movements by Proper Methods of Examination.*—It would therefore appear, from a study of the various factors concerned in compensatory movements of the wrist, that: (1) The forearms should be immobilized to eliminate the factor of momentum, and the movement should be made slowly. (2) The hand should be in a position midway between pronation and supination to eliminate the factor of gravity. (3) Before testing extension, complete relaxation of the opposing muscles should be secured. This is best accomplished by having the patient first flex the wrist, then relax and finally attempt extension. (4) The relative position of the fingers and the hand should remain constant.

What is true of the wrist is also true of other joints, and the same precautionary measures should be taken in all cases to eliminate compensatory movements. In general, the part should be immobilized proximal to the joint, and the movement made slowly. A zero position should be secured to eliminate the factor of gravity, and complete relaxation should be secured in the opposing muscles. No movement in the joints distal to the one under examination should be allowed. Failure to observe these measures when a well marked compensatory movement exists will almost certainly lead, sooner or later, to errors in diagnosis.

IV. LESIONS OF INDIVIDUAL NERVES CONSIDERED IN REFERENCE TO COMPENSATORY MOVEMENTS AND OTHER PHENOMENA

The general principles governing compensatory movements have been discussed. These movements will now be studied in relation to the nerve lesions which have most commonly presented themselves.

A. *Circumflex Nerve Lesions.*—The circumflex nerve is frequently involved in shoulder injuries. In such cases an anesthetic area is found over a portion of the deltoid muscle, and generally the arm can be only slightly abducted from the side unless the scapula is rotated. Occasionally, however, a patient, without apparent contraction in the deltoid muscle, and with everything including sensory and electrical tests indicating a complete lesion of the circumflex nerve, is able to raise the arm to a horizontal position before the scapula commences to rotate, and considerably higher when aided by rotation of this bone. In the cases we studied at Cape May, this movement was due to supraspinatus action. The supraspinatus muscle, however, is not situated advantageously for producing abduction, and without the deltoid the shoulder usually cannot be abducted to any marked degree.

B. *Musculocutaneous Nerve Lesions.*—Musculocutaneous paralysis results in loss of sensation over a portion of the forearm, and weakness in supination and elbow flexion. Although the brachialis anticus and biceps are completely paralyzed, there is not complete loss of any movement of the elbow because the supinator longus, a powerful flexor of the elbow which is innervated by the musculospiral, partially substitutes for the muscles supplied by the musculocutaneous

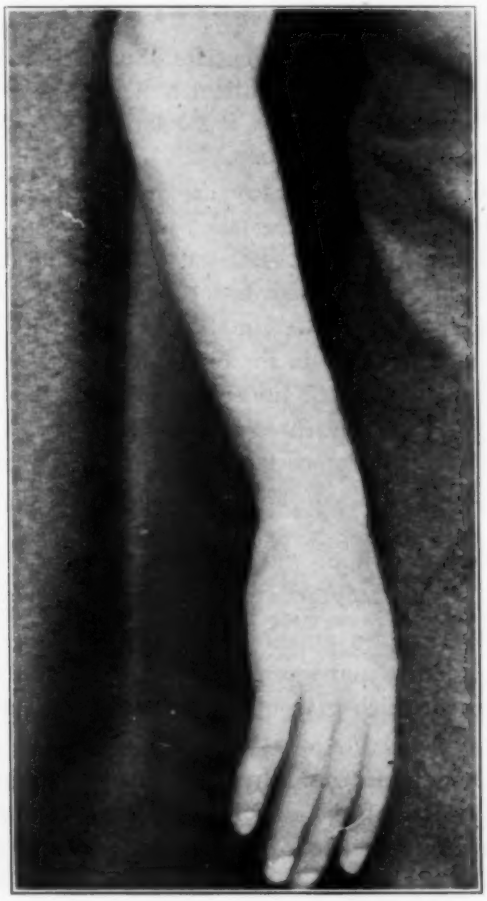

Fig. 5.—Patient with musculospiral and musculocutaneous paralysis with muscles relaxed.

when it is paralyzed. When both the musculocutaneous and the musculospiral are injured, one would expect to find flexion of the elbow and supination abolished. This is often the case. Supination was abolished in all of our cases, but in a few instances flexion of the elbow was obtained through a compensatory movement of contrac-

tion by the pronator radii teres, which is supplied by the median nerve. This muscle, having its origin at the internal condyle of the humerus, and its insertion in the shaft of the radius, is so situated that it does not act with sufficient mechanical advantage to produce strong flexion; but when a patient has trained his power of flexion by exercise, a sufficiently strong contraction may be obtained to flex the elbow to 90 degrees (Fig. 5), and to lead the patient (and perhaps also the physician) to believe that function is returning in the injured nerves. Palpation over the supinator longus and biceps and all other tests at our disposal will, however, show these muscles to be completely paralyzed, whereas the pronator radii teres will be found to contract strongly while the elbow is being flexed. Pronation accompanies the movement. Thus, we find that the musculospiral nerve always partially compensates for the loss of function of the musculocutaneous, and that even when both of these muscles are paralyzed slight compensation is occasionally secured through the pronator radii teres.

C. *Musculospiral Nerve Lesions.*—Loss of musculospiral function results in inability to extend the elbow, wrist and thumb. The metacarpophalangeal joints of the fingers cannot be extended. Besides this there is found, as in the case of musculocutaneous paralysis, weakness of supination and elbow flexion, without complete loss of either, the biceps supplementing the supinator longus and brevis in these movements. It is sometimes difficult to tell whether action in the supinator longus is absent or merely weak, since contraction of the biceps tends to pull on the fascia of the antecubital space, giving an erroneous impression of muscle contraction in the supinator longus. In such cases it is well to fix the forearm by holding the wrist, and then to have the patient attempt to flex the elbow while the palpating finger is applied on the supinator longus, over the shaft of the radius, several centimeters below the insertion of the biceps tendon. At this point any contraction of the supinator longus may readily be discerned.

It is to be noted that in musculospiral paralysis, extension of the interphalangeal joints of the fingers is possible, being accomplished by the interosii and lumbricales (Fig. 6). This movement in some cases is so marked as to simulate musculospiral action. Errors of interpretation from this source may be avoided by carefully observing the metacarpophalangeal joints. No true extension will be found if the case is a complete musculospiral injury.

Low lesions of the musculospiral may be overlooked because of their failure to produce wrist drop. Extension of the wrist is secured by the action of the extensor carpi radialis longior. The branch from the musculospiral to this muscle comes off well above the elbow; therefore a lesion below this point cannot produce wrist drop.

Compensatory movements in musculospiral paralysis in their bearing on the wrist joint have already been discussed, and nothing further need be added. If the examiner is on the alert for these movements and takes the precautions which have been suggested to eliminate compensatory movements of contraction and relaxation, and the factors of gravity and momentum, he should have little difficulty in securing an accurate estimate of the degree of musculospiral loss.

Fig. 6.—Same patient with flexion of the elbow obtained by action of the pronator radii teres.

D. *Median Nerve Lesions.*—Loss of function in the median nerve, besides resulting in loss of sensation over a portion of the hand and fingers, produces loss of thumb flexion and thumb abduction. Next in importance is the marked weakness in flexion of the index finger; there is also weakness in pronation, wrist flexion, apposition of the thumb against any other finger and in flexion of the fingers. No commonly seen peripheral nerve injury affords greater difficulty in diagnosis than does the median, partly on account of the tendency of the

musculospiral and ulnar sensory functions to overlap the median, but particularly on account of the large number of compensatory movements that patients with median paralyses display. When the median nerve is severed, pronation may be begun by the action of the supinator longus and finished by the action of gravity so that an almost normal range of movement is secured without the action of the pronator radii teres and pronator quadratus muscles. Wrist flexion is likewise secured by the synergic action of the flexor carpi ulnaris, extensor ossis metacarpi pollicis and that portion of the flexor profundus digitorum which is supplied by the ulnar nerve. Because of this partial ulnar supply to the flexor profundus digitorum, flexion of the fingers is not lost in median lesions.

Flexion of the thumb may occur through a mechanism of relaxation to which we have already referred, this movement being dependent on the pull of the shortened flexor longus pollicis in the relaxation period following the action of the extensors. Flexion may also be secured by wrist extension when the flexors are shortened. This is another example of a compensatory movement of contraction. Apposition of the thumb to the fingers may be secured by the synergic action of the adductor pollicis, the deep head of the flexor brevis pollicis and the extensors of the thumb.

Abduction of the thumb may be secured in the absence of the abductor pollicis by the combined action of the deep head of the flexor brevis pollicis, which is supplied by the ulnar nerve, and the extensors of the thumb. This movement of substitution is not common, and is generally incomplete, so that loss of thumb abduction is one of the best signs of the absence of median motor function in the hand. A convenient and practical test for this function is to have the patient lay the hand, back down, on a table and attempt to point the thumb straight upward; or the examiner may place the patient's thumb in this position. Inability on the part of the patient to maintain this position of the thumb is evidence of loss of median action.

E. *Ulnar Nerve Lesions.*—Ulnar lesions produce the well-known anesthetic area with a fair degree of constancy. This furnishes an important diagnostic sign, and constitutes a leading factor in the production of the disability which results from such injuries. Destruction of the ulnar nerve results in some cases in surprisingly little motor loss which cannot be compensated for by muscles supplied by other nerves. Generally, the only motion completely absent is abduction and adduction of the fingers.

Flexion of the fingers, with the exception of the thumb, is provided for by the combined action of the median and ulnar nerves. Finger flexion is present, though perhaps weak, in paralysis involving

either one or the other of these nerves. Bearing in mind that the portion of the flexor profundus digitorum, which has its insertion into the little and ring fingers, is supplied by the ulnar nerve alone, one would expect that in ulnar paralysis the terminal phalanges of the little and ring fingers could not be flexed. This is not the case as a rule, because apparently all of the tendons of the deep flexor muscle are acted on to some degree when either the median or the ulnar portion contracts; hence the little and ring fingers are usually flexed in their terminal portions when the index and middle fingers flex. A fairly satisfactory test for function in the ulnar portion of the flexor profundus digitorum is obtained as follows: Completely immobilize the index and middle fingers, and immobilize the little and ring fingers up to the terminal phalanx; then have the patient attempt flexion of the fingers. In the absence of ulnar function, the little and ring fingers cannot be flexed in their terminal phalanges.

In ulnar paralysis the loss of the flexor carpi ulnaris may be compensated for by the action of the extensor carpi ulnaris. This muscle pulls the hand toward the ulnar side, while flexion of the entire wrist is attained through the action of the median flexors of the wrist.

Loss of the lateral movements of the fingers is probably the most distinctive sign of ulnar paralysis. Strangely enough, however, the extensors may be trained to produce lateral movements of the fingers, probably because the extensor tendons do not usually run directly across the apices of the knuckles, or if they do, they are inserted laterally into the phalanges. The action of the lumbricales also tends to produce lateral movement of the fingers. The lateral action which is sometimes found in the little and ring fingers in ulnar paralysis cannot, however, be explained in this manner. A useful method for overcoming this compensatory movement is to put the extensor tendons under tension by flexing the wrist and fingers before attempting the movement of spreading the fingers. Another useful procedure is to palpate the hypothenar eminence while the little finger is abducted. The abductor minimi digiti contracts if ulnar action is present to any marked degree, and the skin over the hypothenar eminence wrinkles and dimples inward as the muscle contracts. The latter action is due to the contraction of the palmaris brevis which is supplied by the ulnar nerve and inserts into the palmar fascia.

F. *Ulnar Combined with Median Nerve Lesions.*—When ulnar and median lesions coexist, flexion of the wrist and fingers is no longer possible except when compensation is secured by means of the factors already discussed. The extensor ossis metacarpi pollicis furnishes the most important action in this respect; a surprising degree of flexion is often obtained by contraction of this muscle (Fig. 7).

Fig. 7.—A patient with complete musculospiral paralysis extending the interphalangeal joints by means of the interosii and lumbricales.

Fig. 8.—Flexion of the wrist by means of the extensor ossis metacarpi pollicis. The case is one of complete ulnar and median paralysis.

G. *Sciatic Nerve Lesions.*—Lesions of the sciatic nerve when complete usually cause loss of all movements below the knee. A striking fact in this connection is that even when the lesion is found to be high on the sciatic nerve, frequently no paralysis of the hamstrings occurs. Another peculiarty of sciatic lesions is the tendencey toward dissociation which they display. Lesions high in the thigh will often produce paralysis only in the internal or external popliteal supply, usually in the latter.

H. *External Popliteal Nerve Lesions.*—In lesions of the external popliteal nerve one finds loss of sensation over the lateral aspect of the foot and leg, inability to evert or extend the foot, and loss of extension in the metacarpophalangeal joints. As in the hand, the distal phalanges can be extended even in the presence of a paralysis of the extensor muscle, the extension being accomplished through the agency of the interosii and lumbricales. All of the types of movements of compensation found in the upper extremity may occur, and this source of error may be overcome by the means already described. Because movements of the toe and ankle joints tend to be more restricted than finger and wrist movements, tendon palpation is a more valuable guide to muscle contraction in the lower extremity than joint action. Before the toes or ankle commence to extend, it is often possible to feel the extensor tendons become taut where they run beneath the anterior annular ligaments of the ankle.

I. *Internal Popliteal Nerve Lesions.*—Internal popliteal lesions are accompanied by anesthesia on the sole of the foot. The motor disturbance consists in loss of plantar flexion of the foot and toes, absence of lateral movements of the toes and inability to evert the foot. A fairly common and somewhat confusing compensatory movement may occur. Some patients obtain plantar flexion of the foot by use of the peronei, which are supplied by the external popliteal nerve. When these muscles contract even when the internal popliteal nerve is completely paralyzed, there may result a diagonal tilting of the foot which may be mistaken for plantar flexion; true plantar flexion may even occur in cases where the peronei insert well back of the axis of rotation of the ankle. Other types of compensatory movements are found to some degree in connection with internal popliteal lesions, but if proper precautions are taken none will be likely to lead to an error in diagnosis with the exception of that to which we have just alluded. This confusing compensatory movement should be suspected if, in the presence of other evidences of internal popliteal paralysis, the patient obtains feeble plantar flexion of the foot without evidence of gastrocnemius contraction. In such cases palpation of the peronei will demonstrate contraction of these muscles, but the Achilles' tendon will be found

to relax instead of tighten with each downward movement of the foot. When these conditions exist, whatever plantar flexion is obtained is the result of a compensatory movement of contraction by the muscles supplied by the external popliteal nerve.

V. CONCLUSION

The list of compensatory and substituted movements might be prolonged indefinitely, but enough have been considered to emphasize the importance of careful observation to avoid errors in diagnosis. In examining the results of a large number of peripheral nerve lesions, the characteristic and classic symptoms will commonly be found; however, a certain number of cases will exhibit unusual phenomena. These atypical cases are the ones that offer the greatest difficulties in neurosurgical diagnosis.

It is hoped that sufficient has been said to show the inadequacy of simply learning a list of the classic symptoms as signs of nerve injuries. The fundamental requisites for accurate diagnosis of such cases include a thorough anatomic knowledge, a mastery of the mechanics of joint action and discriminating observation. With the application of these broad principles to neurosurgical diagnosis, the difficulties are minimized and the proper treatment can be confidently instituted.

A CONSIDERATION OF THE NATURE OF AURAE*

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Descriptive and statistical studies of aurae are to be found in textbooks of nervous and mental disease, treatises on epilepsy and migraine and special articles; but these sources of information are searched almost in vain for an exposition of the particular conditions of the nervous system or of the mind that favor the occurrence of aurae. Such references as one finds generally are bare statements unsupported by argument and obviously taken without question from the conclusions of some distinguished investigator. That these references are scarce and unconvincing is not surprising in view of the difficulty of investigating aurae, as well as other phenomena connected with epilepsy and migraine, first hand in the person afflicted.

The subject would seem to be worthy of consideration, however, because without a proper understanding of the underlying conditions it is hardly possible either to form a satisfactory opinion of the relation of aurae to the convulsions in epilepsy, or to the headaches in migraine; or to make an accurate statistical or analytic study of aurae themselves. In this article, therefore, an attempt is made first, to bring forward objections to what seems to be the prevailing view; second, to point out anew the relation of aurae to a certain mental state, and finally, to suggest some factors that may influence the form the aurae may take.

AURAE OF EPILEPSY

Objections to Prevailing View on Aurae.—The notion of aurae that is most popular with the authors of textbooks probably originated with Hughlings Jackson,[1] but is given very clearly and concisely in these words of Gowers:[2]

"This immediate warning is of the greatest importance . . . The sensation is the effect on consciousness of the commencing process of discharge, which begins in structures by which these are influenced

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*Contribution in a series offered to Prof. E. E. Southard in honor of the decennium of the Bullard Professorship of Neuropathology, Harvard Medical School.

1. Jackson, H.: Brain **11**:179, 1888.

2. Gowers: See Albutt-Rolleston: A System of Medicine, Ed. 2, New York, Macmillan Co. **8**:494.

that are highest in function and related closely to consciousness. The warning is thus an indication of the part of the brain in which discharge begins, because the place where it first attains such an intensity as to cause a sensation, must be the place at which it starts. . . . Their interest is great, but their practical value is almost limited to the detection of attacks which might otherwise be unperceived, by the evidence their character affords, and by their frequent indication that the instability is the result of a local lesion of the brain." There is in this conception of the nature of aurae an application to the sensory component of the epileptic attack of one theory of the origin of convulsions, namely, that which assumes them to be the result of a "discharge" of nervous energy in the motor cells of the precentral area.

Even though this conception of the origin of the convulsive movements of epilepsy be accepted as satisfactory, its application in the explanation of aurae may be objected to. The convulsive movements are of sudden onset and display little evidence of a coordination of nervous processes. On the other hand, the aura may be relatively slow in evolution and if complex in nature, indicates a delicate coordination of nervous impulses that is inconsistent with the notion of a "discharge." Further, the conception is incomplete in that it does not indicate the relation of the aurae which occur in the presence of an organic lesion to those which occur in brains apparently normal in structure. It is begging the question to get over this difficulty by assuming all cases of epilepsy to be the result of an organic brain disease. Structural changes that probably are not secondary are not found in a large proportion of the brains of a certain type of epileptics. In a still larger proportion the aura is not associated with changes in special sense centers that correspond to the special sense in which the aura occurs. It cannot reasonably be stated, then, that the aura of a special sense indicates or may indicate structural or even functional changes of a pathologic nature in the centers of that sense. A theory of aurae should make clear in how far a particular aura may be determined by structural changes and where these changes should be looked for.

In view of the relative futility of efforts to investigate aurae first hand in afflicted persons, it is well to attempt to employ another method, i. e., try to get some light from a study of similar and perhaps less inaccessible phenomena, if such can be found, and to apply the information thus obtained by analogy. There is a mental phenomenon, or rather a series of allied phenomena, of common occurrence in man, namely, hallucinations, to which it would seem aurae could be safely

compared. Parish,[3] McDougal[4] and others have noted the similarity of these manifestations of mental activity. Although some conspicuous differences may immediately stand forth, it is apparent that they are not essentially dissimilar. The subject matter of the one can generally be essentially duplicated in the other. The senses of sight and hearing, moreover, are represented in both in a similar proportion — as 71 to 29 in some statistics of the occurrence of aurae collected from several sources, and as 70 to 30 in the statistics of the occurrence of hallucinations, collected by Parish.[5] The half-recalled memories or "deja vu" phenomena of certain incomplete attacks have many parallels, occurring in connection with sleep and certain waking mental states. No insuperable objection to this comparison is evident, at first sight at least.

Relation of Aurae to Other Hallucinations.—Hallucinations are common in both abnormal and physiologic mental states. Their occurrence in the insane and in those addicted to the use of drugs and alcohol is well known. The most common manifestation in physiologic states occurs in connection with sleep, in the form of dreams. There are also the hallucinations of hypnosis, of crystal gazing and of the psychic medium. Finally, there is the spontaneously occurring variety so much studied by spiritualists, and commonly known as visions.

According to Parish,[6] there is a common mental state underlying all hallucinations, which he designates "dissociation of consciousness." By this term he refers to a state of consciousness that differs from the waking state, in that normal association of ideas does not occur, or in which, as James expresses it, ideas are "deprived of their reductives." Leaving aside as too complicated for consideration the pathologic states in which hallucinations are found, there is the loss of consciousness to a greater or less degree in sleep and a similar, but less profound, loss in hypnosis and crystal gazing, which favors the production of hallucinations. That the psychic medium usually gives her information with eyes closed, would indicate that she may be at such times in a state of autohypnosis. In the case reported by Prince,[7] visions appeared in a prospective medium only after she had gone through a preliminary course of training, which consisted in gazing intently at a small point on a table for a long time. She doubtless was thus acquiring the power of autohypnosis. In the case of the visions reported by various societies for psychical research, Parish was able to demonstrate that in a large

3. Parish: Hallucinations and Illusions, New York, 1909.
4. McDougal: Hallucinations, Encyclopedia Britannica.
5. Parish: Hallucinations and Illusions, New York, 1909, p. 107.
6. Parish: Hallucinations and Illusions, New York, p. 71.
7. Prince: Brain **21**:528, 1898.

proportion they occurred under conditions that favor sleep, or during emotions such as grief, fear or excitement that may produce a somewhat similar dissociation of consciousness. Before reading the conclusions of Parish, I had been impressed with this association merely in going over accounts of visions by various authors.

There is in epilepsy a disturbance of consciousness in the form of a loss. The depth of the unconscious state precludes the recollection of any visions occurring at this time; but there is a brief interval before consciousness is entirely lost that is probably not unlike the normal drowsy state and during which there are probably the conditions which have been shown to be suitable for the development of hallucinations. That hallucinations or aurae do occur at this time is, therefore, not surprising. Hence one should consider aurae as having an origin somewhat similar to that of hallucinations in normal persons, and should not assume, as Gowers and others have assumed, that they result from a "discharge" of an epileptic nature, due perhaps to a local lesion.

Factors Influencing the Form of Aurae.—If this assumption be true, the content of aurae should in general be derived as are dreams and visions. They may represent a fragment of memory, as was the case with one patient of mine whose aura consisted of a familiar face —not always the same face, however—and with another, whose aura was the vision of a disagreeable old woman he had known in childhood. In this connection it is interesting to recall the suggestion of Schwab[8] that aurae may represent memory pictures of events attending the first fit, which have been "fixed," so to speak, by the strong attending emotional reaction. Further, the aura may consist of diminution or distortion of normal perceptions — micropsia, macropsia, etc. — which occurs also in connection with sleep. Finally, the underlying factor may be structural change. Since disease of the organs of special sense or of their pathways leading to the brain may lead to the formation of hallucinations in the corresponding sensory spheres in either the sane or the insane,[9] similar changes may influence aurae. Turner[10] considered these factors, in the case of auditory aurae, without much success; but the subject is worthy of further study.

Some points at which aurae and other varieties of hallucinations differ are worthy of consideration. There is, first, variability; aurae being constant in most cases and hallucinations generally fleeting. This difference may be in part explained by one of the characteristics

8. Schwab: Unpublished contribution presented before St. Louis Neurological Society, 1916.

9. Colman: Brit. M. J. 1894, p. 1015.

10. Turner: Epilepsy, Chicago, Chicago Medical Book Co., 1907, p. 81.

of epilepsy, namely, stereotypy. In general, the various reactions in epilepsy tend to be constant: hence, the preconvulsional disturbance of consciousness should be about the same in all attacks. This being true, the aura would be expected to be relatively constant. On the other hand, drowsy states vary within the widest limits and in them, unlike epileptic mental states, consciousness is often very receptive to external stimuli. It is therefore not surprising that the hallucinations should vary. Another point of difference is the absence of hallucinations during and after the convulsion. The depth of unconsciousness and fatigue factors may account for this lack. Some authors have assumed, however, that hallucinations do occur at this time.

AURAE OF MIGRAINE

As regards migraine, the problem is more obscure. Parallels for some of the aurae of migraine, such as the fortification spectrum described by Gowers for instance, are rare; but as aurae of this type seldom appear, few parallels should be expected. In visions and hallucinations bright lights and brilliantly colored objects are not unusual. Not unlike some migrainous aurae, is the vision of the boy, described by Gurney,[11] who saw in the dark a distant, tiny, luminous point approaching and increasing in size and becoming a face, and finally a mass of luminous faces unlike anything he had ever seen. A patient of mine had, after retiring and before falling asleep, a vision consisting of a "head of flame." That some disturbance of consciousness occurs with migraine there can be little doubt. Migraine is regarded by many as akin to epilepsy, and various abnormal sleep-states are commonly associated with it. One would think that conditions in migraine are even more favorable for the formation of hallucination than are those in epilepsy, inasmuch as in migraine the march of symptoms is slow and consciousness is not lost.

There remains to be considered the relation of the localized brain lesion to aurae. That there is an association of olfactory aurae with certain local lesions Hughlings Jackson,[12] Gowers[13] and Southard[14] have conclusively demonstrated. This fact instead of controverting the view that aurae are merely a type of hallucination, is itself made clear by some characteristics of hallucinations. It already has been pointed out that lesions of organs of special sense or of their pathways leading to the brain, may predispose to aura formation. In the case of the brain lesion, then, it may be assumed that the same principle is at work, the only difference being that interruption of peripheral impulses

11. Gurney: Phantasms of the Living **1**:400, 1886.
12. Jackson: Brain **21**:580, 1898; Jackson and Stewart: Ibid. **22**:534, 1899.
13. Gowers: Brain **32**:303, 1909.
14. Southard: Am. J. Insan. **64**:607, 1907-1908.

occurs in the brain rather than in the peripheral pathways. Indeed, this principle can be applied in instances where another, let us say, that of direct irritation to the cortical cells, cannot be. In a case with olfactory aurae described by Jackson,[12] for instance, the lesion found postmortem was a cyst of softening in the hippocampal gyrus. According to a dictum of Jackson himself, a purely destructive lesion should not be considered as acting as an irritant on surrounding cortical substance. Again, in a case with a similar type of aura, quoted by Gowers,[13] the lesion found at necropsy was a gumma of one olfactory lobe. In neither of these cases, then, can the aura have resulted from an irritation of the olfactory cortex; but both are easily explained as the result in the center of a disturbance of incoming nervous impulses.

CONCLUSION

By way of summary, it may be stated that I have attempted to point out anew the analogy between aurae and the hallucinations occurring in connection with sleep, hypnosis, crystal gazing, etc. According to this view, aurae should be regarded not as the result of "discharges" of an epileptic nature in some part of the cortex, but as deficiency reactions, like dreams, occurring when there is a "disturbance of consciousness" of a certain type. Their relation to the loss or disturbance of consciousness in epilepsy and migraine is assumed to be the same as that of dreams to drowsy or sleep states; and their content should be regarded as being determined by the same factors that determine the content of dreams and similar hallucinations. Their relation to structural changes may be the same as that of those hallucinations which develop in connection with disease of the organs of special sense or of the nerves connecting them with the brain.

A CONTRIBUTION TO THE HISTOPATHOLOGY AND HISTOGENESIS OF SYRINGOMYELIA*

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There still reigns a great confusion of opinions relative to the nature and origin of spinal cord cavities in general, and of those known as syringomyelia in particular. Thus, Schlesinger,[1] after extremely detailed discussion (covering 140 pages) concerning the pathology and pathogenesis of syringomyelia, comes to the conclusion that it may be caused by many anatomically different processes, and offers the following anatomic classification of spinal cord cavities:

1. Cyst formations (caused by injuries of the spinal cord, i. e., traumatic, also nontraumatic hematomyelias).

2. Softenings of inflammatory and noninflammatory origin, with a short course of the morbid process.

3. "Syringomyelien," in the form of: (*a*) malformation; (*b*) an actual tumor formation; (*c*) syringomyelia gliosa; (*d*) syringomyelia from vascular disorders without gliosis, and (*e*) pachymeningitis and leptomeningitis combined with cavity formations.

Schlesinger thus accepts at least five types of syringomyelia, each type differing from the rest anatomically and pathologically. If such be the case, syringomyelia cannot be considered a specific morbid condition, and clinically should be looked on rather as a syndrome. Such a view is shared by practically the greatest majority of students of this disease. For instance, Thomas and Quercy[2] claim that syringomyelia is devoid of any specific anatomopathologic features, and that it would be more proper to substitute the name of medullary cavities for that of syringomyelia, as, in their opinion, there are "syringomyelias" caused by gliomas, developmental anomalies, traumas, hematomyelias, softening, etc.

On the other hand, Phillippe and Oberthür[3] would call the latter types rather pseudosyringomyelias, referring to syringomyelia proper

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*From the pathology laboratories of Cook County and Psychopathic Hospitals of Chicago.

1. Schlesinger, H.: Die Syringomyelie, Ed. 2, Wien und Leipzig, 1902.

2. Thomas and Quercy: Syringomyélie, hyperplasie du tissue conjonctif, fibres musculaires striées dans la moelle, Nouv. iconog. de la Salpêtrière **25**: 364, 1913.

3. Phillippe, C., and Oberthür, J.: Contributions à l'étude de syringomyélie et des autres affections cavitaires de la moelle épinière, Arch de méd. expér. et d'anat. path. **12**:513, 1900.

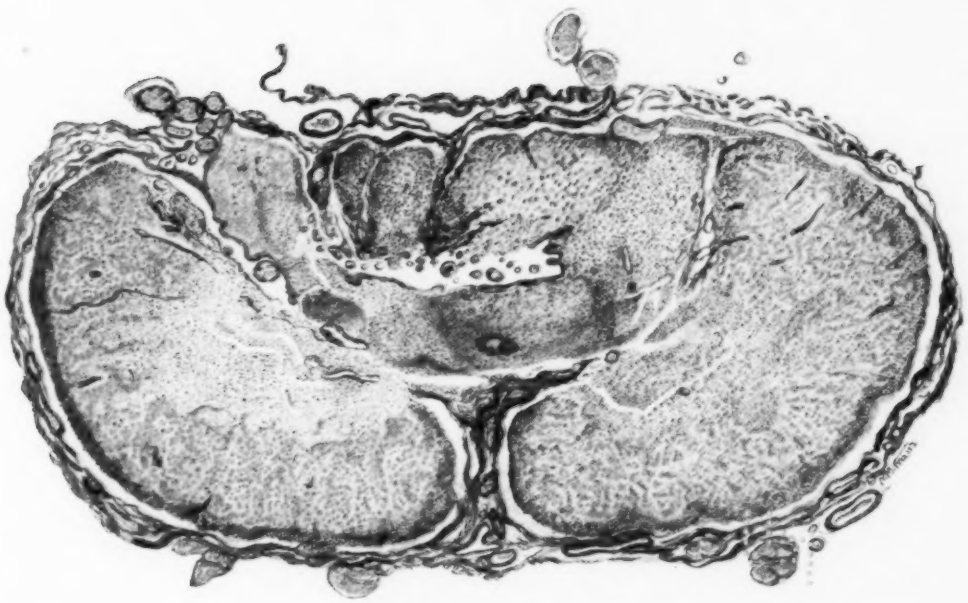

Fig. 9.—Syringomyelia; cervical enlargement. The pia, myelin, axons, gray matter, the pathologic glia, the marginal glia stained in different colors. Combined Bielschowsky-Alzheimer-Mann stain.

Illustrating Article by G. B. Hassin, M.D.,
Archives of Neurology and Psychiatry, February, 1920

only those morbid conditions that possess a certain specific pathology which they claim cannot be confounded with any other morbid condition. Though admitting two types of syringomyelia, cavitary and pachymeningitic, Phillippe and Oberthür believe that they are independent of each other, though caused by the same as yet unknown etiologic factor. The foregoing references represent prevailing opinions relative to the nature of syringomyelia. In spite of an enormous literature bearing on the subject, I believe my findings are of sufficient interest to be recorded.

From the study of my case, as well as from the most important previous contributions to this question, the conclusion may be drawn that in syringomyelia we do possess a number of specific pathologic changes which totally differ from those to be found in any other spinal cord lesion—changes which stamp syringomyelia as a distinct anatomopathologic entity.

REPORT OF CASE

History.—A white, married laborer, aged 54, entered Cook County Hospital, April 26, 1919, on the dermatologic service of Drs. Harris and Stillians, complaining of "sores" on the right arm and left shoulder. About three weeks previously his right arm felt lame, and showed a small, painless, red lump. Two weeks later this lump broke, discharging very foul-smelling pus. The sore on the left shoulder was also painless and of but two days' duration.

He had been a patient at the county hospital thirty-four years before for some trouble with the right eye, which was operated on, but he did not know the nature of the trouble. He had chancre twenty-five years before; and gonorrhea, twenty and two years before. He used to drink. His family history was of no importance.

Examination.—The patient was rather poorly nourished without any deformities, except an opacity over the right pupillary area and scars from an old iridectomy. On the right shoulder there was an ulcer, about 1½ inches in diameter, roughly circular, with a clear-cut margin; the edges were not thickened or everted, exposing the muscles beneath, which formed the floor of the ulcer; there was a scanty, yellowish discharge. The surrounding skin was reddened, scaling and, like the ulcer, painless. Lower down on the same arm, over the deltoid insertion, as well as over the left shoulder, similar ulcers were developing. The rest of the skin, on the back and lower limbs, was covered with "bites," scratch-marks and scars. Over the right hip was a discolored area. The muscles of the arms and both shoulders were atrophied.

Sensibility.—The temperature was abnormal. The pain sense was markedly impaired, the touch less so. The patient was rather indifferent to examination and cooperated poorly; yet it could be demonstrated that the entire surface of the body, including the upper and lower limbs, but excluding the face, was anesthetic, the anesthesia comprising the pain, temperature and touch senses.

Reflexes.—The pupillary (on the left) was sluggish; cremasteric and abdominal, normal; tendon reflexes, markedly exaggerated, with bilateral clonus, positive Babinski, Oppenheim, Gordon and Schaffer. The gait was uncertain, ataxic; the muscle power, greatly diminished. There were no tremors. The

serologic examination gave a negative globulin, and a negative Wassermann test in both the blood and the spinal fluid, 20 cells per cubic milimeter and a negative Lange test (0011210000).

Course.—The ulcers grew larger, spreading over the scapulae, exposing the heads of the humeral bones and copiously discharging pus. Involuntaries (bladder and rectal) set in; the patient became totally paralyzed, apathetic, filthy. The tendon reflexes disappeared entirely; the sensory disorders became more marked, and two months after his entrance into the hospital he died.

Macroscopic Examination of the Spinal Cord and Meninges.—The dura was of normal thickness with smooth, shiny surfaces, without adhesions, pseudomembranes, hemorrhages, or any other pathologic deposits. The pia appeared thickened, especially around the cervicodorsal region and adherent to the spinal

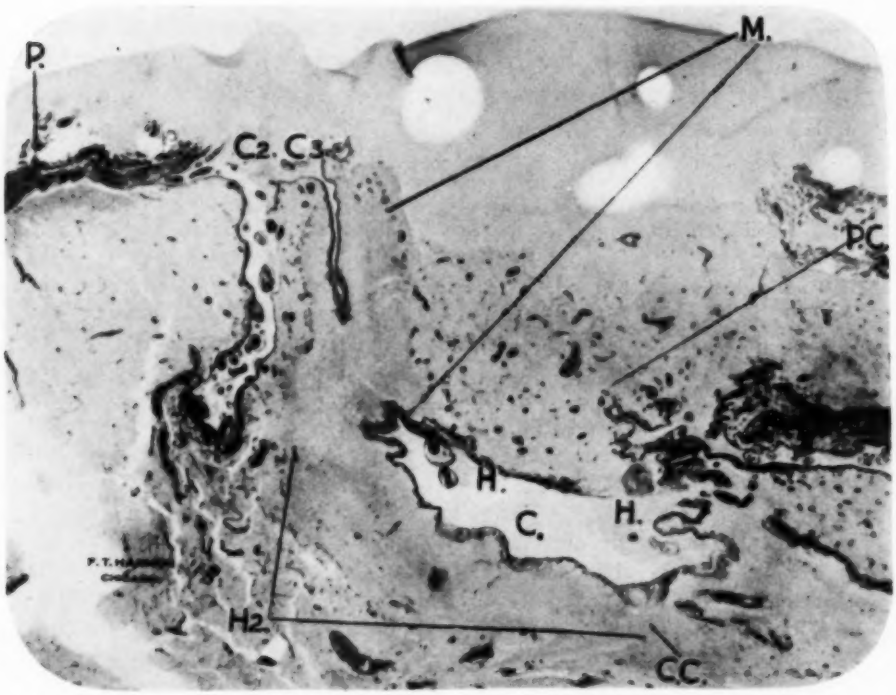

Fig. 1.—Syringomyelia cavities in the cervical cord enlargement. The main cavity (*C.*) contains patches of "homogenized" glia (*H.H.*) and two vascular islands. The accessory cavities (C_2C_3) distinctly show their membranes originating from the pia. H_2 is the zone of "hemogenization;" *P.c.*, posterior columns; *M.*, new forming membrane. Alzheimer-Mann stain; celloidin section $\times 20$.

cord. The latter was greatly flattened in the mid-dorsal region. In the cervical region, which was somewhat enlarged, there was a small cavity, surrounded by a gray area occupying both posterior horns and the greater part of the posterior columns. The upper half of the dorsal portion was very friable, granular in appearance and surrounded by remnants of white matter. The lumbosacral segments were of normal configuration, but on section exhibited

a granular mass in the center, part of which dropped out, leaving irregularly shaped cavities. The lower lumbar region and the entire sacral region presented slightly disfigured posterior columns, being otherwise well preserved.

The medulla, midbrain and the hemispheres revealed nothing pathologic.

Microscopic Examination.—The changes found in the spinal cord in general may be classified as (1) cavities, (2) thickened or hyperplastic pia, (3) a superabundance of vessels and capillaries enclosed within islands, and (4) so-called zones of homogenization.

The cavities showed well only in the cervical region, where six of them could be easily made out. Figure 1 shows three of the cavities; the largest, oblong and very irregular in shape, occupying the middle of the section behind the central canal and lined by a thick connective tissue membrane. The acces-

Fig. 2.—Part of the cervical enlargement of the spinal cord. The center is occupied by a large cavity lined by a collagenous connective tissue membrane. The larger portion of the cavity is filled with pale mass remnants of the homogeneous area surrounding the cavity and separated from the preserved parenchyma of the spinal cord by a thin irregular membrane, *M*. The upper left quadrant is covered with numerous vessels, vascular islands and remnants of a cavity, *C*., lined by a connective tissue membrane. Alzheimer-Mann stain; celloidin section, $\times 23$.

sory cavities, to the left of the larger one, communicate with each other and reach the periphery of the spinal cord, where detachments of the pia can be seen running along their edges and forming a lining. The other three cavities,

not shown in the picture, occupy the right portion of the posterior columns and the right posterior horn. Each cavity contained a powerful connective tissue membrane which did not show any epithelial lining, but was studded in some places with a great amount of vessels (Fig. 1, C_2). The cavities were separated from each other by a homogeneous substance, a pathologic glia, which was also present within the cavities themselves (Figs. 1 and 2). All the cavities ended blindly in the spinal cord and were in no way connected with the central canal, but communicated with the inner surface of the pia. Those portions of the posterior columns which were not taken up by the cavities were converted into a homogeneous mass (Figs. 1 and 2). The anterior horns were cut off by the latter, leaving intact the anterior portion, which was

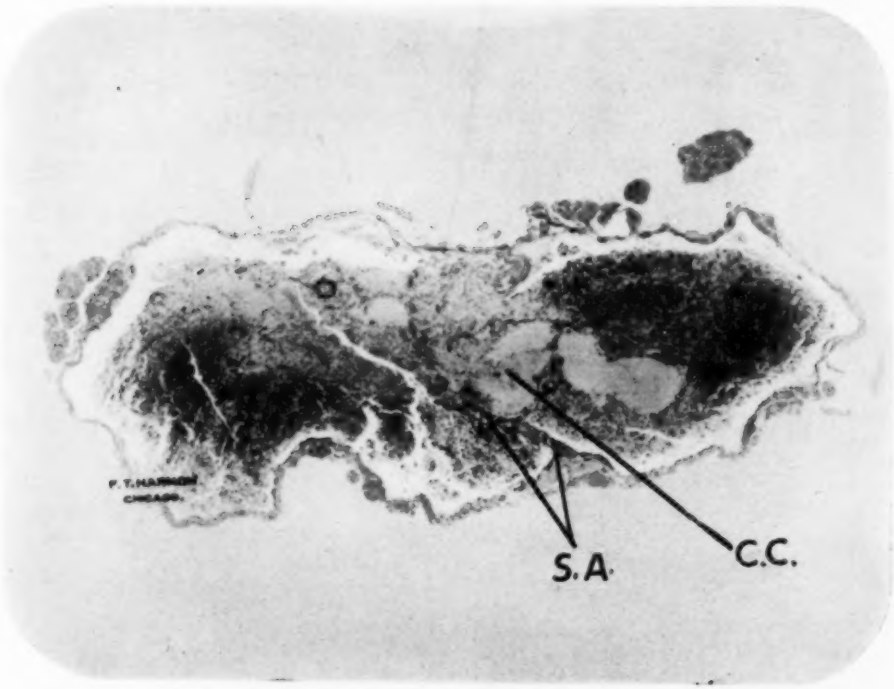

Fig. 3.—Dorsal segment in a case of syringomyelia. The thickened pia is torn off, the parenchyma is collapsed and disfigured. *S.A.*, anterior fissure filled with a thickened pia; *C.C.*, central canal. The lateral columns are well preserved; the broken down posterior half of the spinal cord contains pale islands of pathologic glia. Combined Weigert Pal-Van Gieson stain, × 11.

exceedingly vascular. The lateral and anterior, as well as the undamaged portions of the posterior columns, were more or less well preserved; but many nerve fibers in them were replaced by glia tissue which was much more abundant than in a normal cord. In the dorsal, lumbar and sacral portions there were no real cavities, the empty spaces to be encountered there being due to the falling out of the friable mass. The photomicrograph (Fig. 3) gives some idea of the changes in the dorsal region which was disfigured and collapsed. The lateral and anterior columns were somewhat preserved,

while in the lumbar region (Fig. 4) the posterior columns showed an abundance of connective tissue strands, the subpial marginal glia zone being totally obliterated and the edge of the white matter depressed. Other sections of the dorsal region, partially reproduced in Figure 5, appeared uniform, were granular, and were occupied in some places by connective tissue fibers forming so-called islands. The tissue was very unstable, was friable, and was very losely held together, as if being prevented from falling apart by the surrounding powerful pia. Some portions of the spinal cord substance were missing, and an empty space was plainly in evidence.

In contrast to marked degeneration of the cord substance, the pia was remarkably developed. All along the spinal cord, especially in the cervicodorsal region, this membrane was greatly thickened. It gave a lining to the cavities (Fig. 2), even obliterating some of them and appearing distinctly

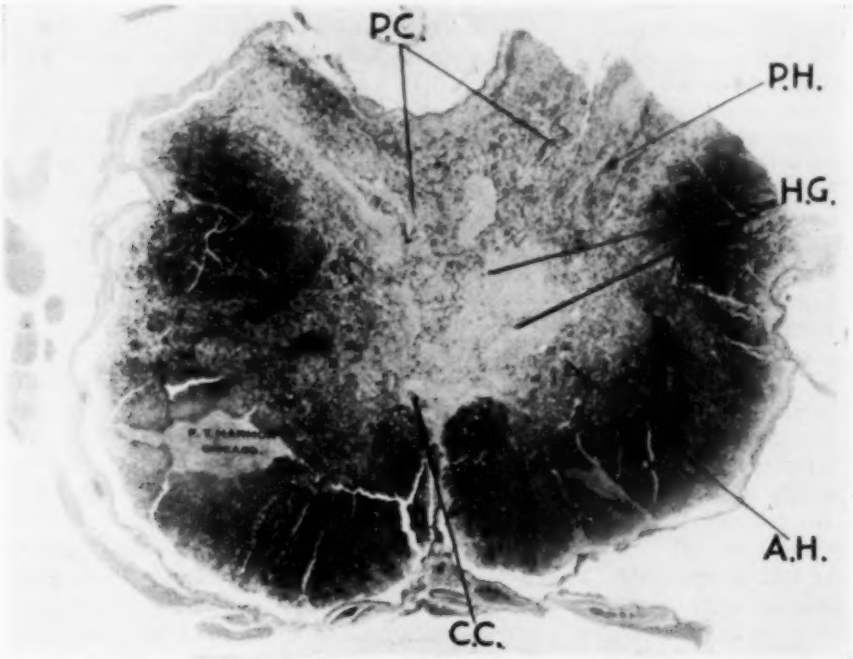

Fig. 4.—Lumbosacral region; the thickened pia forms in the posterior columns a marked concavity; the anterior and lateral columns are well preserved. *C.C.*, central canal; *A.H.*, anterior horn cut off from the posterior horn, *P.H.*, by a mass of homogeneous glia, *H.g.* Weigert-Pal stain counterstained with Van Gieson, × 11.

fibrillar. In thin sections the pia fibers showed no nuclei, but a thicker section showed that the latter could be detected in the form of large spindle-shaped bodies with rounded edges, filled with chromatin granules. Young connective tissue cells, fibroblasts, perivascular infiltrations, plasma cells, lymphocytes, pigment deposits or hemorrhages were totally absent in the pia. The pia entirely enveloped the spinal cord, carrying a great number of hyperemic veins and capillaries. It invaded the spinal cord substance in the form of bands, and

was closely adherent to the subpial glial tissue, but did not press on the posterior or anterior roots. We have here a hyperplastic pia, which, like a fibrous ring, surrounds the spinal cord, especially in places of its greatest destruction (cervicodorsal portions). The outlined features can very well be followed up with Van Gieson, Mallory-Jakob, Alzheimer-Mann, or light-green-fuchsin stains, but especially well with the combined Bielschowsky-Alzheimer method which will be described in detail at the end of this paper.

The vessels were exceedingly numerous in both gray and white substances, in the normal as well as in the pathologic areas. The homogeneous zone surrounding the cavity was rather poor in vessels, but the membrane lining the cavity was as a rule enormously vascularized (Figs. 1 and 6). Yet, even with Weigert's elastic stain, signs of vascular proliferation could not be found. Nor could any signs of inflammation be found, like perivascular infiltrations, distention of Robin-Virchow's lymph spaces in the adventitia, or any intima changes. The lumen was almost always open, containing some red cells, and was narrowed because of the greatly proliferated adventitia. This uniformly showed several layers of connective tissue fibers with few or no nuclei, staining very readily with Van Gieson, Alzheimer-Mann, Jakob-Mallory, and still better with a combined Bielschowsky-Alzheimer-Mann stain. With all these stains it was possible to demonstrate the hyperplastic state of the adventitia in every vessel and capillary to be found in the pathologic area. In the preserved portions of the anterior horns, and lateral and anterior columns, the vessels were hyperemic and in some places were surrounded by a thickened prolongation of the pia, but did not show hyperplastic adventitia. In some regions of the spinal cord, especially the gray matter and around the membranes of the cavities, the vessels, however small, were surrounded by connective tissue rings forming round or oval islands of various sizes. These are the so-called "encapsulations" of Petren[4] (Figs. 5 and 6), already pictured by Lockhart Clarke.[5] These islands or encapsulations sometimes contain several small vessels or capillaries with thickened adventitial walls, consisting, like the other vessels, of hyperplastic connective tissue. The islands were areolar in structure (Fig. 5), and consisted of meshes of glia fibers which were either empty or contained remnants of broken up glia tissue (nuclei, cell bodies and fragments of fibers). In some places the islands showed several concentric rings, or an island came into view that contained several separate rings, these being smaller vessels enveloped by a thick hyperplastic adventitia (Fig. 6). Such sclerosed vessels surrounded by one common capsule were described by Thomas-Hauser[2] as "formations conjonctivo-vasculaires," and by Cerletti[6] "vascular aggregations." Petren calls them vascular islands which he asserts originate from the encapsulations. The capsule comes, he thinks, from the bands of connective tissue invading the posterior horns. They grow so large that for lack of space they have to fold, enveloping portions of gray matter and forming rings enclosing gray matter.

4. Petren, Carl: Beiträge zur pathologische Anatomie und zur Pathogenese der Syringomyelie und der Syringobulbie, Virchows Arch. f. path. anat. **196**: 377, 1909.

5. Clarke, J. Lockhart: On the Pathology of Tetanus, Med. Chir. Trans. **48**:255, 1865; Pathologic Investigations in a Case of Paraplegia, Brit. and Foreign Med. Chir. Rev. **33**:485, 1864.

6. Cerletti, Ugo: Die Gefässvermehrung im Zentralnervensystem; Nissl-Alzheimer's Arb. **3**:1, 1911.

When the latter is absorbed, a vascular island remains. Instead of nerve tissue, we have, then, connective bands and sclerosed vessels—a scar as it were —representing the ultimate healing process of the diseased spinal cord tissue. On the other hand, this connective tissue proliferation accompanied by vascular sclerosis may appear as a forerunner of a progressively developing cavity, acting as a "destructive" agent. The destructive stage is followed by a "constructive," progressing, healing process which takes place after the parenchyma has been destroyed. The connective tissue proliferation is, according to Petren, a chronic inflammation which results in the formation of numerous sclerosed vessels. Such cases Petren calls sclerotic syringomyelia in distinction from the glial type (syringomyelia gliosa) which is due to hyperplasia of the glia. The sclerotic type of syringomyelia runs a shorter clinical

Fig. 5.—Lower dorsal region; the upper portion is granular; the lower, greatly rarefied and in some places devoid of substance. The black dots are cross sections of axons; the paler subtsance is glia tissue. The middle of the illustration is occupied by an "encapsulation" (*Enc.*), the membrane of which is separated from the vascular island (*Vasc. Isl.*) by decomposed glia tissue and remnants of nerve fibers. Bielschowsky stain; paraffin section, × 100.

course; the latter, a more protracted one. Such are some of the conclusions Petren arrived at from the study of connective tissue bands, connective tissue islands and encapsulations.

As these three types were represented in my case, Petren's interpretations are not in complete accord with my findings. The photomicrographs show

the connective tissue formations are present only in those places where there are to be found remnants of a substance described under various names (couches scleroses by Hallopeau,[7] colloid substance by Roth,[8] "Gewebsmasse" by Chiari,[9] homogeneous substance by Schwarz[10] "homogenization" zone by Schlesinger,[1] primitive gliosis by Phillippe and Oberthür[2]). This substance is especially marked in Photomicrograph 2 in the shape of a broad mass surrounding the membrane. This mass can be found within the cavity itself as a patch sticking to the wall, or between two adjacent membranes (Fig. 1) or within the meshes of the latter, but is never found beyond the outer wall of the membrane. The latter thus cuts the glia or the parenchyma into two parts, the inner part, pathologic; the outer, normal.

The microscopic examination of the homogeneous masses showed a network of delicate glia fibers, with a number of small glia nuclei more or less well preserved, though some stained rather badly. Quite often there was but a remnant or a fragment of a glia reticulum with a great mass of uniformly stained, usually pale, round bodies of various sizes. Some of them gave a perfect reaction for amyloid, and there can be no question that many of these round or oval formations were amyloid bodies. Mixed with the latter, there were other spheroid bodies of almost similar size, equally stainable with various anilin dyes, but containing a very small, shrunken nucleus, apparently collapsed, always located at the very periphery of the cell body, as if ready to leave it (Fig. 7). In some such cells half of the nucleus could be seen as if sticking out of the cytoplasm, and some cells were encountered without any nucleus whatever. Such cells greatly resemble amyloid bodies, from which they differ by failing to show the iodin reaction. It is probable that some such cells ultimately become actual amyloid bodies, in accordance with the views of Fromman, Obersteiner, Redlich[11] and others who consider them as the products or modifications of glia. In addition, there is in the homogeneous substance a third variety of spherical globules, very numerous and much smaller and paler, appearing as fine, minute droplets. They are especially numerous within the cavities, in the walled-off, or as Thomas and Hauser[12] express it, in the areas "parceled out" by connective tissue bands and vessels. They are also to be found in great masses within the connective tissue spaces, in the adventitial walls, in the encapsulations or islands, in the pia meshes, in short, wherever the glia is destroyed or damaged. One gains the impression that the proliferating connective tissue in spreading to the periphery envelops them as a useless substance, before they finally perish and become absorbed. The remarkable feature is that even in these greatly changed patches of glia tissue, there may be found well preserved nerve fibers, or,

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7. Hallopeau, H.: Contributions à l'étude de la sclérose diffuse péri-ependymaire, Soc. de Biologie, Ser. 5, **21**:169, 1869.

8. Roth, W.: Gliome diffuse de la moelle, syringomyélie, atrophie musculaire, Arch. de physiol. normale et pathol. **10**:612, 1878.

9. Chiari.: Ueber die Pathogenese der sogenanten Syringomyelie, Ztschr. f. Heilk. **9**:307, 1888.

10. Schwarz: Ein Fall von Meningo-myelitis Syphilitica mit Höhlenbildung im Rückenmark, Ztschr. f. klin. Med. **34**:467, 1898.

11. Redlich, E.: Die Amyloidkörperchen des Nervensystems, Jahrb. f. Psychiat. u. Neurol. **10**:1, 1891.

12. Thomas, A., and Hauser, G.: Histologie pathologique et pathogénie de la syringomyélie, Nouv. iconogr. de la Salpêtrière **17**:376, 1904.

what is more frequent, fragments of well-stained myelin and axons. These changes in the "homogenized" area can be much better followed up in longitudinal section (Fig. 7). The photomicrograph distinctly shows the nerve fibers, enlarged or ampule-like, in some instances thinned, of distinct fibrillary structure and staining well. In some fibers the axon is plainly visible; in others, it is masked by a powerful myelin sheath forming basket-like enlargements connected with each other by thin strands of fibrils. The spaces between adjacent fibers is occupied by the round bodies and spherules described above, the entire mass resembling Alzheimer's "Füllkörperchen" and his ameboid glia. Places less damaged or not much disintegrated show a few poorly stained gitter-cells, large protoplasm-rich glia cells uniformly stained, with an eccentric

Fig. 6.—Vascular islands, *V.I.;* the rest of the round formations are encapsulations, some inclosing longitudinally cut vessels; from an area near the cavity. Combined Bielschowsky-Alzheimer-Mann stain; paraffin section, × 85.

nucleus, and occasionally even so-called myelophages. Herxheimer stain, which was hard to carry out on account of the difficulty of obtaining proper frozen sections of a friable granular mass, showed the presence of fat globules.

The central canal was found occluded by a mass of proliferated cells, nowhere connected with the cavities, and surrounded by a homogeneous mass which microscopically (Fig. 8) appeared to consist of a delicate glia reticulum containing a number of glia nuclei. It somewhat resembled the structure of the "homogenization" zone, but the process evidently had not progressed very far. The lateral and anterior columns and the preserved gray matter of the

anterior horns showed no gross abnormalities, like ganglion cell changes, secondary degenerations, etc. The medulla and cortex exhibited vascular changes only. In the former, some vessels were slightly infiltrated with lymphocytes. In the cortex there was marked proliferation of capillaries, without any other changes, like infiltrations, ganglion, glia or meningeal changes. Pathologic changes were exclusively confined to the spinal cord, being at some levels more pronounced, at others less, confined principally to the glia, the vessels and pia, with or without cavity formation. It is obvious that the latter is not an essential factor in syringomyelia, as many levels in this case did not show cavities at all. Probably they would have formed had the patient lived

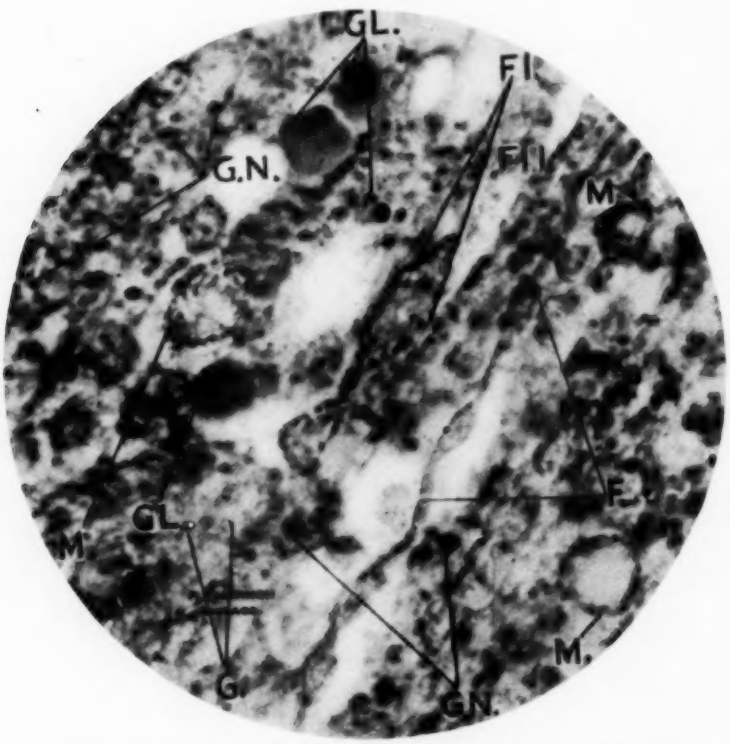

Fig. 7.—Longitudinal section of the "zone of homogenization." *G.n.*, glia nuclei; *Gl*, glia cells with picnotic nuclei about to desert the homogeneous cell bodies; *M*, myelin; *Fi*, nerve fiber with granular ampular enlargements of the myelin sheath; *F* and *Fii* comparatively less changed nerve fibers; *G*, sale glia cells resembling amyloid bodies. Combined Bielschowsky-Alzheimer-Mann stain, × 700.

longer; that is, the changed areas which appeared as homogeneous zones would ultimately have become transformed into cavities.

Of the five factors (changes in the pia, the vessels, areas of homogenization, island and membrane formation (Petren's encapsulations), the most important, the primary factor, is change in the glia. But the character and nature of these glia changes are quite different from those to be found in

various other spinal cord lesions, like multiple sclerosis, secondary degenerations, myelitis, amyotrophic lateral sclerosis and subacute combined cord degeneration, in which glia changes are also the dominant feature. For in syringomyelia the glia does not show the exquisite progressive or proliferative properties so pronounced in the former diseases, nor the equally exquisite regressive character seen in myelitis. It is true that in syringomyelia there may be encountered some signs of a proliferative nature, represented by a few gitter-cells, fat globules, few myelophages, and large protoplasmic glia cells; but these signs are so insignificant, so scarce, and so overshadowed by the opposite phenomena of retrogressive character, outlined above, that at least in this case we cannot speak of a proliferation of glia.

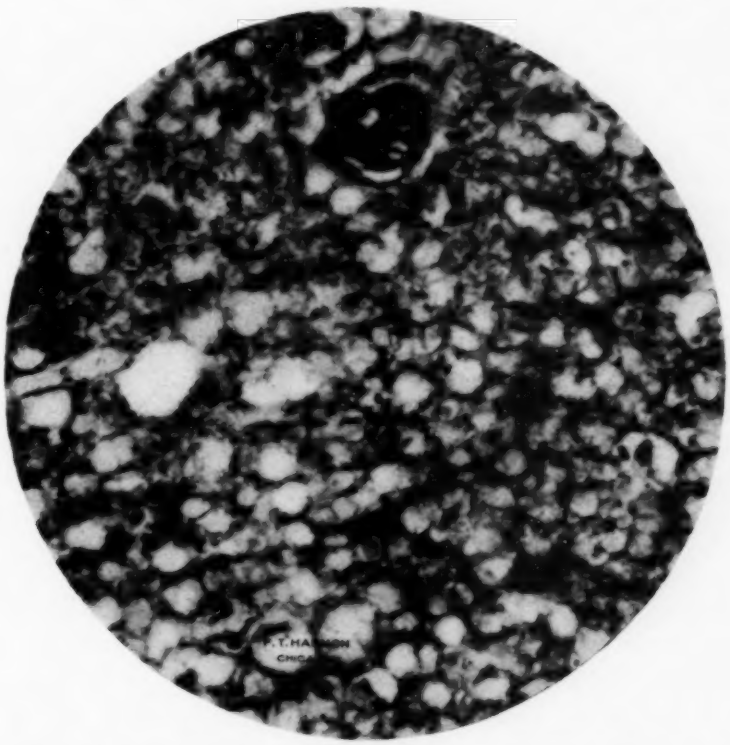

Fig. 8.—An area from the posterior commissure consisting of a glia reticulum, glia nuclei (black dots) and an aggregation of vessels (in the upper portion of the illustration). Combined Bielschowsky-Alzheimer-Mann stain; paraffin section, × 450.

On the contrary, the glia is rather regressive in character, especially near the cavities where the process must be assumed to be greatly advanced, while in the lower dorsal regions the morbid process is evidently of more recent date, no cavity formation being present. Furthermore, if glia tissue does proliferate, as in multiple sclerosis, subacute combined degeneration or secondary degenerations caused by cerebral lesions (capsular or encephalitic lesions), the proliferation obtains at the expense of destroyed nerve tissue. The latter,

however, is in syringomyelia, as shown by my case, not degenerated. Even in the most damaged areas, like the interior of the cavities themselves, nerve fiber fragments with their myelin and axon can be seen comparatively undamaged; that is, not enveloped by the glia cells, nor converted into lipoid substances, harbored by numerous and various gitter-cells, as is the case when the glia undergoes real proliferation. Nor are the glia changes in syringomyelia of the type seen in myelitis, for instance, where it assumes the ameboid features.

COMMENT

Evidently the syringomyelic changes of glia are altogether different from those to be found in progressive or regressive states, a fact already noted by Schultze[13] in 1882, when he coined for these glia changes in syringomyelia a new name, that of gliosis. Ten years later, Hoffman[14] called it primary, central, gliosis. To make clear the meaning of gliosis, Schultze defined it as the mildest or milder stage of glioma formation, as the forerunner of gliosis. In a later publication[15] he compared its relationship to glioma as that of "a hill to a mountain." Bernstein and Horwith[16] express themselves still more indefinitely. "There are seen," they say, "areas of gliosis which perhaps may bear toward the tissue we call glioma a relation similar to that which a fibroma bears to sarcoma (leaving out of consideration the question of malignancy)." Thus, according to Schultze and others, the proliferated glia in syringomyelia is different from a proliferated glia in other spinal cord diseases. If glia proliferates and breaks down it is called gliosis; if it proliferates and does not break down (multiple sclerosis, for instance) then it is not a gliosis. According to modern neuropathologic research, such a distinction between the two types of a proliferative glia is wholly untenable; in fact, it is incomprehensible. From Schultze's publications and his numerous followers (Miura,[17] for instance) the sole outstanding, undoubted fact remains that in syringomyelia we have to deal with a primary lesion of glia, either around the central canal, or in Rolando's substance, or in some other part of the spinal cord. This fact is absolutely established, and we therefore must come to the conclusion that syringomyelia is essentially a disease of the glia and not of the nerve tissue, like multiple sclerosis,

13. Schultze, L.: Beiträge zur Pathologie und pathologische Anatomie des centralen Nervensystems, Virchows Arch. f. path. Anat. **87**:510, 1882.

14. Hoffman, J.: Zur Lehre von der Lehre von der Syringomyelie, Deutsch. Ztschr. f. Nervenh. **3**:1, 1892.

15. Schultze, F.: Die Pathogenese der Syringomyelie, Compt. rend. du 12me Congrés Int. de Med. à Moscou **4**:265, 1897.

16. Bernstein, E. P., and Horwith, S.: Syringomyelia with Pathological Findings, Med. Rec. **84**:698, 1913.

17. Miura, K.: Ueber Gliom des Rückenmarks und Syringomyelie, Beit. z. path. Anat. u. z. allg. Path. (Ziegler's Beitr.) **11**:91, 1892.

amyotrophic lateral sclerosis, subacute combined degeneration, etc., which are essentially parenchymatous nerve lesions, in which the glia is secondarily involved.

The glia changes must necessarily be exceedingly chronic in course, as many cases last from forty to fifty years. In fact, the beginning of the trouble can never be definitely asserted, as the patient seeks advice only when he is much discomforted by some motor, trophic or other disturbance, and may go around for years without complaining, yet harboring the disease. This was probably the case with my patient, who gave a history of but three weeks' duration, and for some symptoms, two days. The deepest and most advanced destruction was in the dorsal spinal cord which suggests the possibility of his having some objective sensory disturbances on the trunk, chest, lumbar region, etc., which did not trouble him in any way until they became complicated by trophic lesions and forced him to go to the hospital. The process in every case must be much older than the date of the patient's first complaints, probably extending, as Saxer[18] justly remarks, to the embryonal state, and probably being inborn. In other words, a person suffering from syringomyelia is probably born with a deficient glia, just as a person with progressive muscular atrophy or dystrophy is born with deficient ganglion cells or deficient muscle fibers; in both we have to deal with a developmental defect, an abiotrophy. The abiotrophy, in the former, shows itself in glia cells; in the latter, in the nerve or muscle cells.

As in the latter a great many ganglion cells are usually undamaged, appearing healthy, so in the former (syringomyelia) a great many glia cells may escape destruction and may even exhibit signs of increased activity, in the form of a rich cytoplasm, formation of myelophages, gitter-cells, etc. As in progressive muscular atrophy, in which the deficient ganglion cells may be confined to certain levels or segments of the spinal cord, the cervical region, for instance, in syringomyelia, the deficient glia also may be confined to a certain level, sparing the rest. When the deficient glia finally breaks down, disintegrates, it becomes a useless foreign body, which like any other foreign body becomes enveloped or walled off by connective tissue. The connective tissue proliferation is thus, contrary to the assertions of Hauser, Sterling[19] and others, a secondary process, obtaining only when glia destruction is at its maximum, originating, as my case shows, in the pia and partly from the vessels. It is of hyperplastic, prolifera-

18. Saxer, F.: Beit. z. path. Anat. u. z. allg. Path. (Ziegler's Beitr.) **32**: 276, 1902.

19. Sterling, W.: Beiträge zur Lehre von der Morvanschen Krankheit, Ztschr. f. klin. Med. **56**:474, 1905.

tive, not inflammatory character, and may be looked on as a natural reaction to the pathologic process displayed in the cavity. The latter is filled with fluid, and is usually greatly distended, the distention of course affecting the pia greatly.

I had an opportunity to witness such a distention during an operation (by Dr. Karl Meyer) on a patient with syringomyelia symptoms, which I had reason to believe were probably due to an intraspinal tumor. On opening the dura in the lower cervical region, the cord appeared distended, sausage-like, very pale, and without any signs of pulsation. When the posterior columns were incised, fluid came out with great force; the spinal cord collapsed at once, became hyperemic, and immediately showed active pulsations. The enormous pressure sustained by the cord in syringomyelia must be counterbalanced by the surrounding meninges, especially the pia, which is called on to hold the fluid and the remnants of the broken up cord firmly together, the hyperplasia being the ultimate result of this increased function. The same is true of the blood vessels, the adventitial walls of which are always greatly thickened, hyperplastic, thus protecting them from a too increased pressure and a probable total occlusion. This vascular state is absolutely pathognomonic of syringomyelia, as in no other pathologic state could I see anything like it, and it is also pointed out without exception in every description of syringomyelia. Very often the sclerosed vessel is the center of various islands, encapsulations, which, as I have already stated, resemble in their structure, mode of growth, etc., the "homogenization" zone around the cavity.

If the glia is deficient from earliest life, then apparently normal spinal cords must be met which would show an abnormal glia state in the vicinity of the central canal or in the posterior horns, the usual seats of syringomyelia. This is actually the fact. Thus Schlesinger and Obersteiner[20] saw many spinal cords, otherwise normal, showing a wider and larger posterior commissure, protruding into the posterior columns in the form of bands or massive processes and consisting of glia meshes, vessels and even occasional areas of homogenization or of fully developed gliosis. Zappert[21] found in a spinal cord of a 19-months-old child a dilated spinal canal, glia proliferation, in which a homogeneous zone surrounded the central canal. These facts demonstrate the correctness of the assumption that syringomyelia dates back to the earliest days of existence and may run through the entire life unobserved or undiscovered; in short, that syringomyelia is a developmental anomaly. But this developmental anomaly does not pertain

20. Schlesinger, H.: Die Syringomyelie, Ed. 2, 1902, p. 324.

21. Zappert, J.: Kinderrückenmark und Syringomyelie, Wien. klin. Wchnschr. **14**:949, 1901.

to a patent central canal (Virchow,[22] Leyden[23]) or to a broken down tumor (Simon,[24] Westphal[25]) or to slumbering epithelial cells around a maloccluded central canal which suddenly wake up and proliferate (Hoffman[14]), or to a proliferation and subsequent breaking down of glia cells (Schultze[13, 15]), but pertains to the inborn deficiency of glia tissue as outlined above. Nor has syringomyelia anything to do with traumatic and nontraumatic hematomyelias (Minor,[26] Pitres and Sabrazes[27]), myelitis (Hallopeau,[7] Joffroy-Achard[28]), pachymeningitis or leptomeningitis (Rosenblath[29]), all these morbid conditions having been advocated as the possible causes of syringomyelia. They all may produce cavities in the spinal cord, but not syringomyelia, which differs from the enumerated morbid states anatomically and histologically. Only those spinal cavities should be referred to as syringomyelia which exhibit glia or other changes as represented, in part, by the appended photomicrographs.

STAINING METHODS

The staining methods must necessarily vary according to the variety of tissues encountered in this case; axons, myelin, glia tissue, gray matter, vessels and pia. Of the numerous methods used, the most reliable and satisfactory one proved to be that of Bielschowsky counterstained with Mann's mixture (methyl blue-eosin). I had used it very succesfully on frozen sections in my studies of subacute combined cord degenerations,[30] while in this case frozen sections could not be used very well on account of the great disintegration of the tissues. I was therefore compelled to use paraffin sections of tissues that were fixed in chrom-fluorine and potassium bichromate (Weigert's

22. Virchow, R.: Die Betheiligung des Rückenmarks an der Spina Bifida und die Hydromyelie, Virchows Arch. f. path. Anat. **27**:575, 1863.

23. Leyden, E.: Klinik der Rückenmarkskrankheiten, **2**:447, 1875.

24. Simon, T.: Beiträge zur Pathologie und pathologische Anatomie des Centralnervensystems, Arch. f. Psychiat. **5**:108, 1875.

25. Westphal, C.: Ueber einen Fall von Höhlen-und-Geschwulstbildung im Rückenmark mit Erkrankung des verlängerten Markes, Arch. f. Psychiat. **5**: 90, 1875.

26. Minor, L.: Zur Lehre von der Syringomyelie, Ztschr. f. klin. Med. **34**: 373, 1898.

27. Pitres and Sabrazès: Contributions a l'étude clinique et anatomie pathologique de l'hematomyélie centrale, Compt. rend. du Congrés Int. à Moscou **1**:292, 1897.

28. Joffroy, A., and Achard: De la myélite cavitaire, Arch. de Physiol. normale et pathol. **10**:435, 1887.

29. Rosenblath, W.: Zur Casuistik der Syringomyelie und Pachymeningitis cervicalis hypertrophica, Deutsch. Arch. für klin. Med. **51**:210, 1893.

30. Hassin, G. B.: Histologic findings in two cases of subacute combined cord degeneration, Med. Rec. **91**:885, 1917.

mordant fluid). After removal of paraffin the sections are stained with Bielschowsky's method in the usual manner. Without being impregnated with gold they are counterstained with Alzheimer-Mann (1 per cent. phospho-molybdic acid) for one hour, followed, after the sections are twice washed in distilled water, by methyl-blue-eosin for one half hour or longer. The pictures are strikingly beautiful, the connective tissue, the glia, the axons, myelin, the gray matter, all staining in different colors as shown in the appended colored reproduction.

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ANALYSES OF BLOOD OF INSANE PATIENTS

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Ishida[1] reported a deficiency of chlorin in the blood of dementia praecox patients and suggested the use of sodium chlorid solution intravenously in the treatment of this condition. He estimated the chlorin values of the blood of five untreated cases of dementia praecox, and the amounts varied from 0.199 to 0.220 per cent. The analyses were made by Professor Horinchi by the Vollhard-Salkowski method. Guthrie[2] published a report of the results of the treatment of fifteen cases of dementia praecox based on chemical analyses of the blood and reported a deficiency of chlorin, but the quantities found and the methods of analysis are not mentioned. Rappleye[3] stated that in some cases of catatonia he found a deficiency of chlorin. Miller[4] quoted analyses made by Weston of the blood of ten cases of dementia praecox, the chlorin being normal in each case, and he stated that "there was no deficiency of chlorin in the blood . . . in any of fifteen other cases examined." Rappleye[3] studied the blood urea in catatonic patients at from three to seven day intervals before, during and after the catatonic state. He found that a marked drop, about 50 per cent., in the urea nitrogen occurred early and rapidly in catatonia.

The figures of Ishida, Rappleye and Weston do not agree. In Rappleye's cases the urea was determined when catatonia was present, as well as when it was not. Weston's figures are from one analysis made when the patient was not in the catatonic state.

Since there is some disagreement in the published estimates of chlorin and urea nitrogen, the accompanying tables may be of interest. They represent analyses of the blood of thirty insane patients, selected cases of dementia praecox, epilepsy and manic-depressive insanity. The analyses were made a little more than two years ago. All the patients were living under practically identical conditions and had substantially the same diet. As the blood was collected before breakfast, twelve hours after the preceding meal, the effect of diet may be disregarded. Care was taken to use clear cases, and in each case selected there was unanimity of opinion when the patient was pre-

1. Ishida, N.: Am. J. Insan. **73**:541 (Jan.) 1917.
2. Guthrie: Dementia Praecox Studies **1**:61, 1918.
3. Rappleye, W. C.: J. Nerv. & Ment. Dis. **49**:130 (Feb.) 1919.
4. Miller, W. C.: Arch. Neurol. & Psychiat. **1**:313 (March) 1919.

sented at a staff meeting for diagnosis. The Wassermann reaction in all cases was negative (cholesterolized human heart antigen), and no abnormal substances were found in the urine of any patient.

TABLE 1.—Blood Analyses of Ten Cases of Manic Depressive Insanity

Number	Age	Total Nitrogen, Percentage	Milligrams per Hundred C.c. of Blood								Remarks
			Nonprotein Nitrogen	Uric Acid	Urea Nitrogen	Creatinin	Creatinin plus Creatin	Glucose	Chlorin as Sodium Chlorid	Calcium	
1	33	3.85	36	2.12	24	2.15	8.90	90	480	8.0	Hypomanic
2	40	3.45	33	2.60	16	1.90	7.60	90	490	7.8	Hypomanic
3	38	3.60	34	1.95	22	2.10	8.40	100	500	7.0	Hypomanic
4	29	3.55	33	2.20	20	1.85	7.20	110	510	9.1	Depressed
5	40	3.20	31	2.40	20	1.75	7.60	120	506	8.8	Depressed
6	36	3.45	33	1.80	24	1.60	7.40	90	490	7.7	Depressed
7	29	3.50	33	2.20	27	2.00	8.00	85	484	8.8	Depressed
8	37	3.70	35	2.10	24	2.10	7.90	90	480	8.4	Interval
9	40	3.60	34	2.30	22	1.90	8.00	90	490	8.0	Interval
10	44	3.75	32	2.75	20	1.80	7.00	110	510	7.8	Interval

TABLE 2.—Blood Analyses of Ten Epilepsy Cases

Number	Age	Total Nitrogen, Percentage	Milligrams per Hundred C.c. of Blood								Remarks
			Nonprotein Nitrogen	Uric Acid	Urea Nitrogen	Creatinin	Creatinin plus Creatin	Glucose	Chlorin as Sodium Chlorid	Calcium	
11	35	3.16	32	1.90	18	1.25	6.20	100	480	7.8	Grand mal
12	32	3.52	36	1.85	20	2.25	8.20	90	495	7.8	Grand mal
13	28	3.48	34	2.20	22	2.31	9.40	110	506	8.8	Grand mal
14	31	3.21	37	2.16	20	1.95	7.60	105	512	8.0	Grand mal
15	31	3.75	36	2.30	21	1.70	8.40	121	500	7.7	Grand mal
16	35	3.64	32	2.00	22	1.67	7.80	95	484	7.0	Grand mal
17	40	3.80	33	1.85	24	2.00	8.10	90	510	7.5	Grand mal
18	37	3.72	34	1.90	20	2.20	8.20	110	490	6.9	Grand mal
19	22	3.69	32	1.76	21	2.12	8.00	120	486	7.6	Grand mal
20	28	3.71	38	2.00	24	2.10	7.95	100	495	7.9	Grand mal

No seizures were recorded for forty-eight hours preceding the analyses.

TECHNIC

Blood was first drawn for the usual routine Wassermann reaction, and then at least 30 c.c. were collected in a tube containing 0.45 gm. of powdered, calcium-free sodium citrate. Oxalate was not used because calcium determinations were to be made with the blood. Analysis was begun at once, and four specimens were examined at the same time, this being as large a number as could be handled without an assistant.

The blood was divided as follows:

Ten c.c. of blood were added to 30 c.c. of 6.25 per cent. trichloracetic acid solution (freshly prepared) in a 50 c.c. volumetric flask.

The mixture was well shaken, distilled water was added until the solution reached the mark, and the flask was set aside for one hour. At the end of this time, the contents were poured into a tube and centrifugalized. The supernatant fluid was pipetted off or filtered through a double-washed filter paper and used for the determination of chorids by titration with potassium thiocyanate, nonprotein nitrogen by the method of Folin and Denis,[5] and calcium by Lyman's method.[6] The use of trichloracetic acid did not affect the chlorin values. Blanks were run each day for calcium and chlorin.

TABLE 3.—BLOOD ANALYSES OF TEN DEMENTIA PRAECOX CASES

Number	Age	Total Nitrogen, Percentage	Milligrams per Hundred C.c. of Blood								Remarks
			Nonprotein Nitrogen	Uric Acid	Urea Nitrogen	Creatinin	Creatinin plus Creatin	Glucose	Chlorin as Sodium Chlorid	Calcium	
21	21	3.52	31	2.40	16	2.00	8.10	105	515	5.7	Hebephrenic
22	18	3.75	33	2.60	17	1.82	7.90	101	510	5.5	Hebephrenic
23	28	3.44	34	1.50	22	1.90	8.00	106	510	6.2	Hebephrenic
24	21	3.26	33	1.90	19	2.16	7.20	111	494	8.0	Catatonic
25	36	3.64	31	2.00	16	2.20	10.00	120	491	8.0	Catatonic
26	27	4.00	39	2.60	22	1.85	9.40	112	506	7.8	Catatonic
27	23	4.00	40	2.70	18	2.10	8.10	105	512	7.8	Catatonic
28	26	3.80	33	1.90	16	1.90	8.66	124	500	6.0	Paranoid
29	29	3.25	33	2.00	16	1.70	9.00	117	500	8.0	Paranoid
30	32	3.19	33	1.80	18	2.15	9.21	109	515	6.5	Paranoid

Blood was not drawn during catatonia.

TABLE 4.—NORMAL VALUES *

	Author		
	Gettler and Baker[8]	Folin and Denis[19]	Myers and Fine[20]
Total nitrogen	3.7	.....	3.0
Nonprotein nitrogen	30 - 45	22 - 37	25 - 30
Uric acid	1 - 3.5	0.7 - 3.7	1 - 2
Urea nitrogen	15 - 25	12 - 27	12 - 15
Creatinin	0.1 - 0.5	1.1 - 1.4	1 - 2
Creatin	3 - 6.5	5.2 - 8.1	5 - 10
Glucose	50 - 120	90 - 100[14]	90 - 130[15]
Chlorin as sodium chlorid	450 - 500	490[16]	457[17]
Calcium as Ca	5.2 - 9.6[18]		

* Data for this table were taken from the paper of Gettler and Baker. Total nitrogen is expressed in per cent. All other values are in terms of milligrams per hundred c.c. of blood. The figures for calcium are from Lyman's paper.

Five c.c. of blood were used for the determination of creatin, creatinin and glucose by the methods of Myers and Fine.[7]

5. Folin, O., and Denis, W.: J. Biol. Chem. **26**:473 (Sept.) 1916.

6. Lyman, H.: J. Biol. Chem. **29**:169 (March) 1917.

7. Myers, V. C., and Fine, M. S.: Chemical Composition of the Blood in Health and Disease, Post-Graduate **30**:39, 1915.

One c.c. of blood was used for the urea determination, and one c.c. for control. Dunning urease tablets were used for fermentation, and the ammonia was determined by Nesslerization after aeration.

One c.c. of blood was added to 99 c.c. of ammonia-free distilled water and 1 c.c. of the dilution used for the determination of total nitrogen by the method of Folin and Denis.[5]

The remaining 10 or more cubic centimeters were used for the determination of uric acid by the method of Myers and Fine.[7]

The Kober colorimeter and nephelometer were used throughout and all glassware was certified.

The creatinin values recorded in the tables are far above the normals given by Gettler and Baker,[8] and Gettler.[9] For the method employed, however, the values are not above normal. The picric acid used answered the requirements laid down by Folin and Doisy,[10] and the sodium citrate, in the quantities used, had no bleaching effect.

Cholesterol is not included in the tables. Several hundred estimations have been made, but the results did not vary from the normal. Some of the results have already been published.[11]

The relation of the sugar content of the blood and spinal fluid was reported some years ago.[12] In sixty-five cases, the spinal fluid sugar averaged 60 per cent. of the blood sugar. Recently, Myers and Fine[13] found in fifteen cases that the spinal fluid sugar averaged 57 per cent. of that in the blood.

SUMMARY

The blood of epileptic, dementia praecox and manic-depressive patients shows no deviation from the normal content of total nitrogen, nonprotein nitrogen, uric acid, urea, creatinin, creatin, glucose, chlorin or calcium.[14]

8. Gettler, A. O., and Baker, W.: J. Biol. Chem. **25**:211 (June) 1916.
9. Gettler, A. O.: J. Biol. Chem. **29**:47 (Feb.) 1917.
10. Folin, O., and Doisy, E. A.: J. Biol. Chem. **28**:349 (Jan.) 1917.
11. Weston and Kent: J. M. Research **26**:531, 1912.
12. Weston, P. G.: J. M. Research **35**:199 (Nov.) 1916.
13. Myers, V. C., and Fine, M. S.: J. Biol. Chem. **37**:239 (Feb.) 1919.
14. In addition to the references given, the following papers may be of interest: Lewis and Benedict: J. Biol. Chem. **20**:61, 1915; Michaelis: Biochem. Ztschr. **59**:211, 1916; McLean and Vanslyke: J. Biol. Chem. **21**:361, 1915; Rogee and Fritsch: Biochem. Ztschr. **54**:53, 1913; Lyman, H.: J. Biol. Chem. **30**:1 (May) 1917; Folin and Denis: J. Biol. Chem. **14**:29, 1913; Myers, V. C., and Fine, M. S.: Post-Graduate, New York **29**:473, 1914.

SPLINTS USED FOR PERIPHERAL NERVE CASES AT THE U. S. ARMY GENERAL HOSPITAL NO. 11

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BOISE, IDAHO

Shortly after patients from overseas began to arrive at the U. S. Army General Hospital No. 11, it was decided that the splints which had been applied to the nerve cases had numerous disadvantages. They were heavy and cumbersome, and in a large number of cases were retarding rather than aiding recovery. With these faults in mind, each lesion was studied from the standpoint of splints, and a special group of splints was designed for each type of case. These were then tried out and the one found, by actual practice, to be the most satisfactory was adopted as a standard splint for a given lesion. The following descriptions, pictures and diagram cover the splints now in use.

SPLINT FOR FOOT DROP

This splint has been adopted as a standard splint for patients who are able to wear shoes, for it is sightly, simple and servicable. It is sightly because the bronze spring wire running along the sole of the shoe blends with the russet leather, while the spiral leggings completely hide the upper part of the splint. They also give added security. It is simple because it is made of two pieces of wire weighing but 2⅓ ounces, and when applied has no point of uncomfortable pressure or of constriction. It is serviceable because it not only holds the foot well extended at all times, but also aids in walking, as it gives the foot the normal flexibility which is lacking in all of these cases.

Pieces of bronzed spring steel wire, 25 inches long, have loops (¾ of an inch long) bent on each end, in opposite directions (Diag. 1). The lower loop is for aid in attaching the wire around the shoe, while that at the top protects the clothing from the sharp wire.

To give the splint flexibility, the wire has a 1¾ turn coil ¾ inch in diameter, bent into it 6 inches from the lower end (Diag. 2). This coil, when in place, is directly opposite the junction of the sole and heel of the shoe.

Two wires, bent as described above, are held in place, one on either side of the shoe, by four pieces of fine piano wire; one piece under the instep, another around the heel at the junction of the upper and heel; a third under the sole, connecting the lower extremities of the wire; and lastly, a piece around the toe between the sole and the upper. The last piece binds the splint permanently to the shoe. It is best to apply the wires in the above order (Diag. 3).

Fig. 1.—Splint for foot drop made of No. 11 spring steel wire and held to the shoe with four pieces of piano wire. The splint is held to the leg by a canvas band passing around the calf between the two wires.

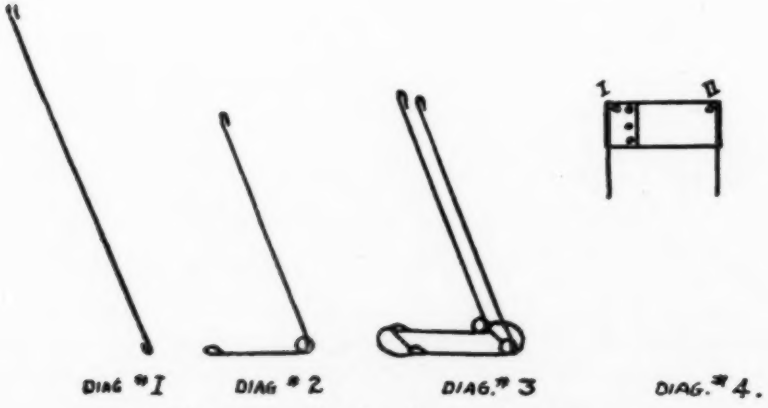

Fig. 2.—Details of splint for foot drop.

The splint is completed by a piece of double ply canvas, 6 inches wide and 4 inches long, and held together by five rivets placed as shown in Diagram 4. Rivets I and II keep the splint from slipping through the canvas. The canvas is slipped over the upper end of the splint, around the calf, and the whole is held in place with a spiral legging.

It takes thirty minutes to make and attach the splint, and it lasts from two to three months.

SPLINT FOR CASES OF WRIST DROP

A wrist drop splint, which is 100 per cent. effective, must perform five definite and distinct functions. First, it must extend the hand on the wrist. Second, it must extend the proximal phalanges on the metacarpal bones. Theoretically, all of the phalanges should be extended, but by actual measurement the small amount of relaxation thus

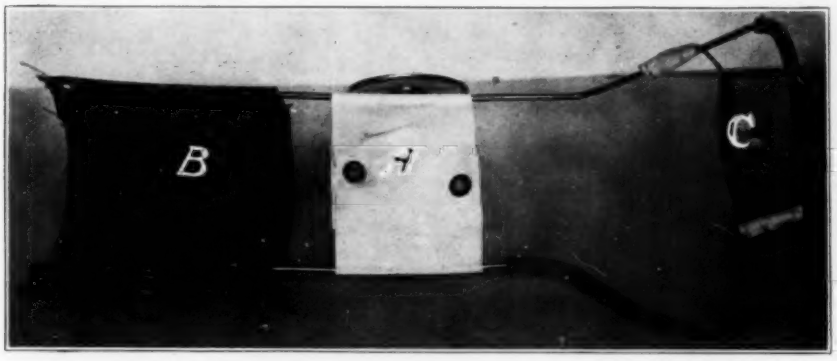

Fig. 3.—Splint for wrist drop with extra piece to support palmar arch. A, adhesive band which fits on dorsal surface of wrist; B, removable canvas band which fits on anterior surface of forearm; C, palmar piece to maintain palmar arch held to the main splint by adhesive plaster.

obtained was decided to be less important than the maintenance of normal mobility of the distal joints by permitting free movement. Third, it must extend the thumb, and by extension is meant a position in a plane with the other fingers. Fourth, it should maintain the palmar arch. Finally, it should be light, simple and comfortable, and should give the part as much freedom of motion as is desirable.

The old aphorism that multiplicity of methods proves lack of a satisfactory one, certainly holds true for the splints used in cases of wrist drop. In the past few months no less than sixteen different types of splint, from home and abroad, have found their way into this hospital. According to our standards, none of them have been even theoretically 100 per cent. efficient, and but few attain over 40 per cent.

Fig. 4.—Wrist splint drop applied. Thumb is held extended in a plane with the rest of the fingers; distal two phalanges are left free so as to permit normal movement. No bandages are necessary to hold the splint in place.

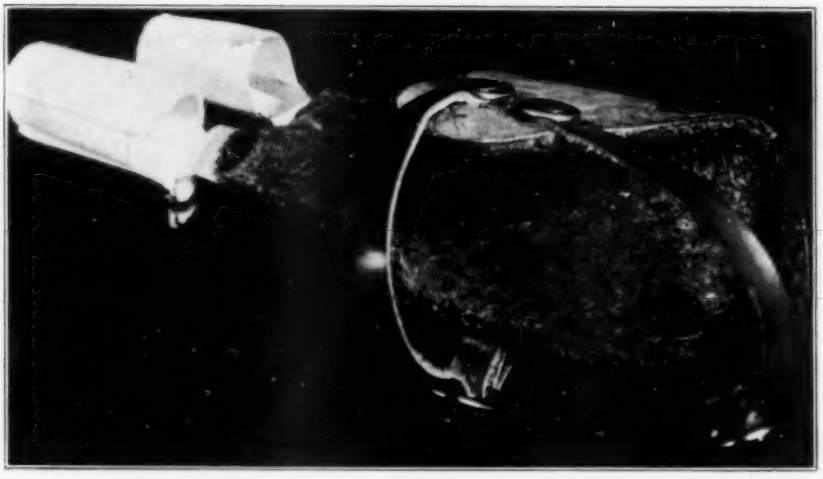

Fig. 5.—Splint for ulnar paralysis. It is made of galvanized iron, padded with felt. The felt is stuck to the iron with sealing wax. Four copper rivets hold the straps in place. The whole is then fitted to the ulnar side of the hand. A forward bend of the finger extensions corrects the tendency to hyperextension. The finger pieces made of galvanized iron padded and surrounded with adhesive tape are held to the main splint with rubber bands which produce the required extension.

Spring steel wire covered with rubber tubing at all points of pressure and properly bent has been found to make a light and comfortable splint. It will meet the requirements and overcome some of the objections of other types. This splint is made from a piece of No. 11 spring steel wire, 35 inches long.

It is best to start bending the splint on the ulnar side of the forearm. Nine inches from the end, the wire is bent upward to form an angle of 140 degrees. This is the angle at which the hand is extended on the wrist. From this point the wire extends forward to the junction of the proximal and middle phalanges of the little finger, where it is bent at a right angle in the same plane. Three quarters of an inch

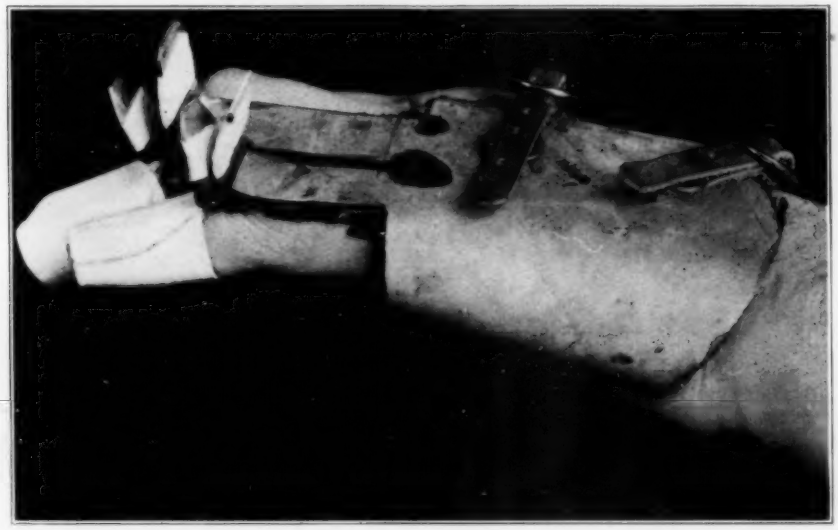

Fig. 6.—Splint for ulnar paralysis applied.

from here it again receives a right angle bend, but this time in a plane at right angles to the previous one. This bend causes the wire to run across the palmar surface of the fingers and thereby extends the proximal phalanges on the metacarpal bones. A right angle bend in the same plane, at the outer side of the first finger, and three quarters of an inch farther on another one in a plane at right angles to it completes that part of the splint which keeps the hand in place. The wire then runs backward along the outer side of the first finger until it reaches a point opposite the base of the distal phalanx of the thumb, where it is bent outward at right angles to extend the thumb on the first metacarpal bone. When it reaches the outer side of the thumb the wire is curved downward and inward to the wrist, following the

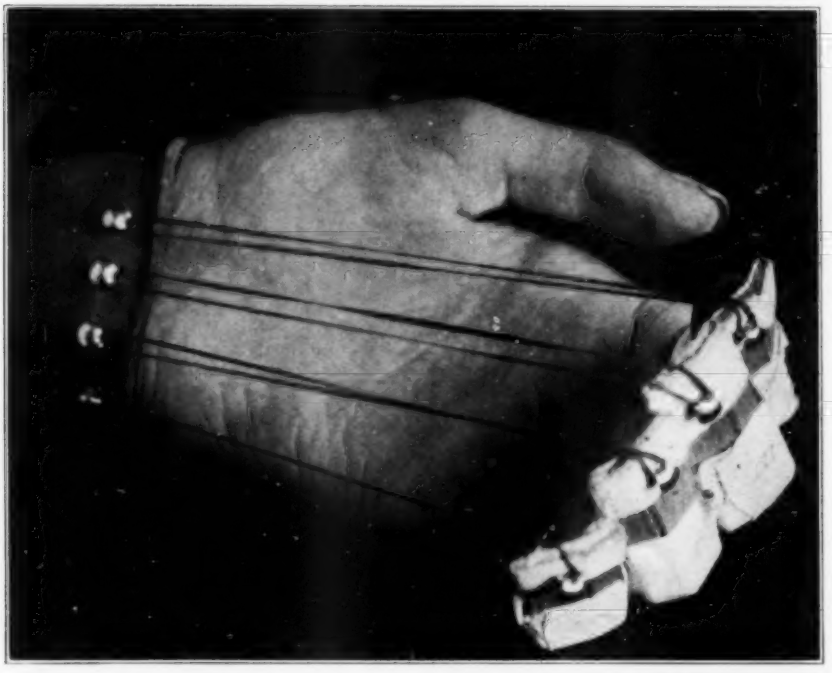

Fig. 7.—First position of twelve hour splints for cases of fixation of the fingers. The splint is worn in this position for twelve hours out of the twenty-four.

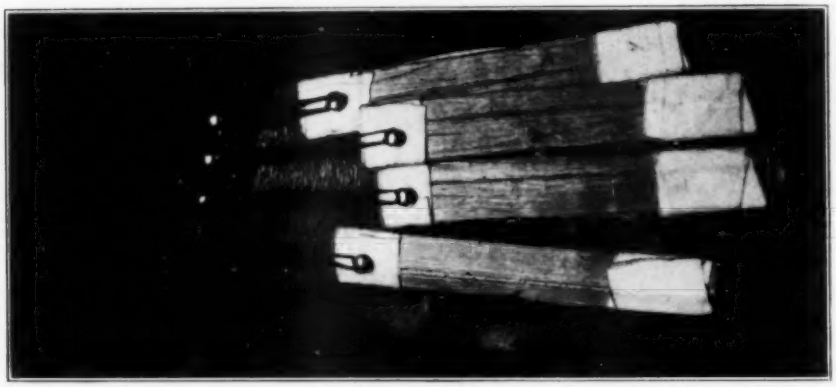

Fig. 8.—Second position of twelve hour splints.

thumb as nearly as possible in its extended position. The wire is now bent so that it runs parallel to and in the same plane as the wire on the ulnar side of the forearm. The two rubber tubes which protect both the fingers and the thumb from the splint are now slipped into place, and then the ends of the wire are bent on themselves, thus protecting the patient from the sharp ends. The palmar arch support is made from No. 20 spring steel wire, covered with rubber tubing, and is attached to the splint by adhesive plaster. The ends are one inch long and are bent in opposite directions from each other to increase the stability of the palmar attachment. The wire first extends downward away from the splint for an inch on each side and then by a slow curve extends upward to support the palmar arch. A piece of adhesive plaster is now placed on the splint just above the wrist and folded on itself so that all of the sticking surface is covered. Finally, the canvas

Fig. 9.—Splint for median paralysis applied. Finger pieces are made of thin board held together by strips of adhesive tape.

band is slipped over the ends coming in contact with the flexor surface of the forearm.

This splint can be made in fifteen minutes. It is applied without bandages and can be readily removed or put on by the patient, without assistance.

SPLINT FOR ULNAR PARALYSIS

A very satisfactory ulnar splint, which not only aids in extending the fingers but also allows them free motion, is made from a piece of galvanized iron cut from a pattern, but shaped to the hand of each individual so that it closely grasps the ulnar half of the hand. It is held in place by two straps, one just below the hypothenar eminence and the other just above the web between the thumb and the first

finger. To give additional comfort, the splint is lined with blanket material stuck to the galvanized iron with sealing wax. The forward projections are of such length that when one half inch is turned up the projections extend just beyond the first phalangeal joint. Another piece of galvanized iron one half inch wide and one and one half inches long is bent at right angles so that one half inch of it corresponds to the half inch upward bend on the projection of the main splint. Around the remaining one inch adhesive tape is wrapped so as to form a cap for the end of the finger, the inner part of this cap being lined so that it can be removed or applied at will. A rubber band binding the two upright extensions together completes the splint.

Fig. 10.—Splint for brachial plexus cases applied. It transfers the weight to the opposite shoulder and to the thigh of the same side. The splint proper is made from 3/16 iron. The canvas straps are held in place with eyelets.

TWELVE HOUR SPLINT FOR BRACHIAL PLEXUS CASES IN WHICH THE FINGERS HAVE STIFFENED IN SEMIFLEXION

Wooden coaptation splint material is used. Short strips are cut, the ends being protected by adhesive plaster. Rings of adhesive plaster, lined so that they do not stick to the skin, are stuck to one end of this splint and fitted to the fingers. A shoe hook is set in the other end of the splint. The shoe hook is then fastened by a rubber band to a corresponding shoe hook placed in a canvas wrist band.

The patient is supplied with a long and a short set of these splints, and is told to wear them interchangeably — the short set to produce flexion and the longer ones to produce extension.

SPLINT FOR MEDIAN NERVE CASES

The splint for the median nerve case (inability to flex the thumb and fingers) differs in no way from the flexion service used in the twelve hour splint, except that it includes the thumb.

SPLINT FOR CIRCUMFLEX NERVE CASES

This splint was copied from one sent from the Lakewood Hospital. It is made from $\frac{3}{16}$ inch iron, and is much lighter and very much less disagreeable to wear than the old aeroplane splint formerly used for these cases. The points of pressure in this splint are the sound shoulder and the hip of the affected side.

410 Overland Building.

A CASE OF MENINGO-ENCEPHALITIS (LETHARGIC ENCEPHALITIS)*

WILLIAM W. HALA, M.D. AND CYRIL M. SMITH, M.D.

BROOKLYN

The purpose of this article is to report a case, clinically diagnosed as encephalitis lethargica, verified by observations antemortem and postmortem.

As a result of the influenza epidemic which raged in Southern and Middle Europe in 1890, cases of epidemic lethargy appeared in Italy and in Hungary.[1] Economo,[2] writing about an epidemic which occurred in Vienna in 1917, likewise speaks of these cases, which at the time were called "nona." He introduced the term encephalitis lethargica. Wilson[3] gives a description of the pathologic changes in the brain of cases of this disease and ascribes the cause to a toxi-infective origin.

Von Weissner, as mentioned in the special article on lethargic encephalitis in *The Journal of the American Medical Association*, March 15, 1919, recovered a gram-positive diplostreptococcus which he claims produced somnolence when injected into apes. Up to the present time, however, no organism has been described which could be held as the etiologic cause of this disease.

Among the recent literature in this country, O. L. Pothier[4] made a report of eight cases occurring at Camp Lee with necropsy findings in one case. Bossoe[5] of Chicago then reported a series of twelve cases observed by him, with two necropsies. The histologic changes that he observed are very definite and agree substantially with those observed in our case. Finally, Tucker,[6] in a recent article, calls attention to eleven cases, with necropsy findings in two of them.

The case reported in this paper is interesting because it displayed not only the characteristc histopathologic lesions observed in the brain by others, but also on account of the intense purulent meningitis and

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* From the pathological Laboratory, Kings County Hospital.

1. Netter: Lethargic Encephalitis, Paris méd. **8**:81 (Aug. 3) 1918; Bull. de l'Acad. de méd., Par. **79**:337 (May 7) 1918.

2. Economo: Verein f. Psychiat. u. Neur. in Wien, April 17, 1917.

3. Wilson, S. A. K.: Lethargic Encephalitis, Lancet **2**:7 (July 6) 1918.

4. Pothier, O. L.: Lethargic Encephalitis, J. A. M. A. **72**:715 (March 8) 1919.

5. Bossoe, P.: Epidemic Encephalitis, J. A. M. A. **72**:971 (April 5) 1919.

6. Tucker, B. R.: Epidemic Encephalitis Lethargica, J. A. M. A. **72**:1448 (May 17) 1919.

ependymitis found at necropsy. The fact that an organism was isolated both antemortem and postmortem, which evidently was the etiologic factor concerned, is noteworthy.

Vaughan,[7] in an excellent résumé, has discussed the disease from a clinicopathologic standpoint in a recent issue of the *Journal of Laboratory and Clinical Medicine*.

Dr. John H. Larkin[8] has informed us of two cases of hemorrhagic myelomalacia that he has observed both clinically and at necropsy, in which the histopathologic changes in the cord were similar to those present in the brain of our case. These cases are to be reported shortly.

REPORT OF CASE

History.—Our case is that of a male, aged 31, a bottler, who was admitted to the general medical service of Drs. Hall and Merrill in Kings County Hospital, Brooklyn, March 24, 1919. The family history was negative except that his wife had died of influenza two months previously. His first visit to a physician was on March 14, at which time his ailment was diagnosed as influenza. The attack was not sufficiently severe to confine him to bed continuously. Two days prior to admission he collapsed suddenly, and from that time he appeared somewhat drowsy.

His chief complaint on admission was malaise, headache and fever. He appeared dull and drowsy, and his responses to questions were slow and retarded. General physical examination was negative except that the neck and back were somewhat rigid, yet the neck could be turned without manifestations of pain. Kernig's sign was absent, and other reflexes, deep and superficial, were not exaggerated. Tactile sensation was present.

Symptomatology.—Examination made on the day following admission showed increasing rigidity of the back and cervical region, but not sufficiently severe to produce opisthotonos. No pain was evinced on active rotation of the neck, but limitation of movement of the head on the shoulder, most marked on the right side, was present. The pupils were dilated and sluggish. His face assumed a mask-like appearance, and he responded to questions though seemingly not understanding. He was not excitable. Superficial and deep reflexes were present and apparently normal; no tâche cérébrale. He swallowed liquids without difficulty.

On March 27 the patient's condition was slightly improved, but he maintained the drowsy appearance and listless attitude. The eyes made feeble attempts to follow moving objects. At times respiration was noisy but never labored. Eye disturbances were negative except for a mucopurulent discharge from the palpebral conjunctiva. A slight bilateral ptosis was present, evidencing a disturbance of the nerve supply in the muscularis palpebrarum. The tongue was dry and parched, and the face muscles on the left were apparently weakened allowing the mouth to deviate to the right.

On March 28 the pupils reacted to light and accommodation, the right being slightly irregular in contour. Efforts to arouse him were futile, but he would

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7. Vaughan: Encephalitis Lethargica, J. Lab. & Clin. M. **4**:381 (April) 1919.

8. Larkin, J. H., Director Strecker Memorial Laboratory, City Hospital, New York. Oral communication.

make an attempt to protrude his tongue after repeated commands, and he would try to grip hands. Examination of the superficial and deep reflexes showed a sluggish right abdominal reflex; right achilles present and sluggish; no Babinski reflex, Chaddock's, Oppenheim's or Westphal's sign.

On March 29 both pupils reacted similarly to the day previously, though not so sluggish. The ptosis was more pronounced. Urinary incontinence developed.

On March 30 the deep and superficial reflexes had entirely disappeared; the body was more rigid. The pupils were irregular in contour and immobile. Horizontal nystagmoid movements were pronounced, and there was evidence of a partial paralysis of the left external rectus. The disks were edematous, but not atrophied. The retinal vessels were congested and tortuous. On account of urinary retention, catheterization was necessary, and obstinate constipation yielded to enemas. Feeding by stomach tube was instituted because of the increasing dysphagia. Toward night increased difficulty in breathing developed, associated with a tendency to pulmonary edema. The semicomatose condition of the previous days had assumed a lethargic character. The tongue deviated to the right. There was increased paralysis of the left facial muscles with a jaw clonus, rapid and regular in rate.

On March 31 the rigidity of the body had entirely disappeared. The pupils were small and immobile. Complete paralysis of the left external rectus of the eye and of the muscles of deglutition were evident. Toward evening pulmonary edema increased, and the patient died at midnight.

Clinical Course.—On admission, his temperature was 104 F.; pulse 96, regular and of good volume; respiration 28. During the next three days the temperature varied from 101 to 103.2, reaching 105 at 8 a. m., March 29. From that time until death the temperature maintained a fairly normal level at 104 F. There does not appear to be any relation between the pulse rate and the temperature (Fig. 1).

Laboratory Findings.—A cytologic examination of the spinal fluid the first day showed a total of 480 cells per c.mm., with a differential count of eighty-four polymorphonuclears, and sixteen lymphocytes; Noguchi globulin test +; no bacteria found in smear; culture negative. Subsequent cytologic findings on the 28th, 400 cells, 88 per cent. polys, 12 per cent. lymphs, globulin +; culture, negative; smear, negative.

Wassermann test negative on blood and spinal fluid. Lange's test on two occasions was as follows: Before administration of 40 c.c. Flexner's antimeningococcic serum 0 0 0 0 1 3 2 1 0; after serum 0 0 0 0 1 1 1 0 0.

Urine, the specific gravity was 1.024, amber, acid, no sugar, trace of albumin, 3.5 per cent. urea, 1.2 per cent. creatinin, trace of mucus, hyaline casts, few epithelial and white blood cells.

Widal test on 29th with 1:20 dilution was negative, blood urea 34 mgm. per 100 c.c., blood sugar, 0.12 per cent.

Spinal punctures were done with the following results: 25th, 2 c.c., negative; 26th, fluid clear, increased pressure, no débris or cloudiness; 27th, dry tap; 28th, no change in character; 29th, no change in character, culture again negative and smear negative; 30th, fluid slightly cloudy, under pressure; cell count 12,000 per c.cm., 90 per cent. polys, 10 per cent. lymphs. Examination of smear showed a motile bacillus, gram-negative, staining readily with ordinary anilin dyes.

GRAPHIC CHART

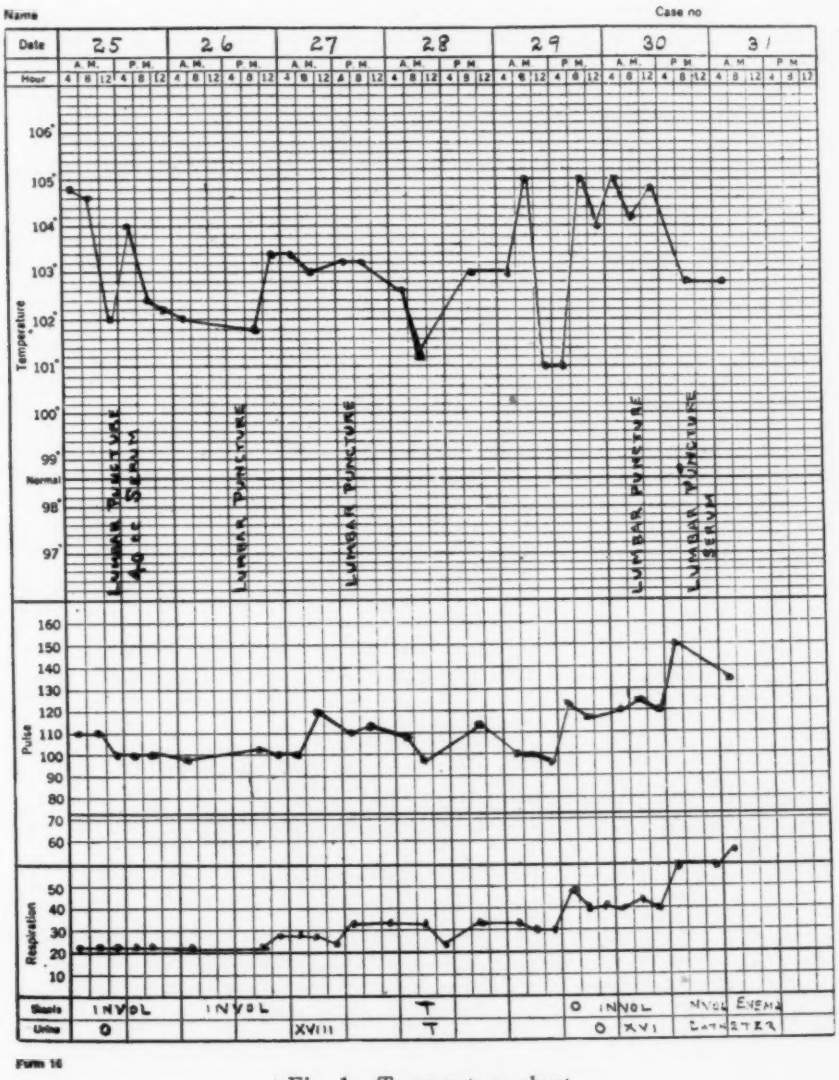

Fig. 1.—Temperature chart.

Differential and total white blood count: March 27, 12,000, 76 per cent. polys, 24 per cent. lymphs; March 21, 24,000, 85 per cent. polys, 15 per cent. lymphs.

Necropsy (performed April 1, 1919).—Necropsy on the body shows the following: Congestion and edema and lobular pneumonia of lungs; septic spleen; parenchymatous degeneration of the liver; acute nephrosis of left kidney; cystic remains of right kidney with anomaly of ureter; subdural hemorrhage; acute exudative meningitis of brain and cord; acute encephalitis; acute ependymitis; petechial hemorrhage in corpus striatum, centrum ovale and optic thalamus, right side.

Head: On removing the calvarium we found the dura adherent over the entire right hemisphere. The brain was therefore removed with the dura attached. On reflecting back the dura of the left hemisphere, the entire cerebral cortex was injected, the pial vessels being rather prominent in places. Small hemorrhagic areas were seen in the pia, and in the sulci between the convolutions a purulent exudate was seen, especially along the fissure of Rolando. On the right side the adherent dura apparently incorporated an oval, rather firm tumor, compressing the motor area of the right cerebral cortex. Incision into this dural tumor disclosed an encapsulated mass, brownish red in color and soft in consistency, suggestive of subdural bloodclot. Observation of the base of the brain, particularly of the pia of the cerebellum, pons and surrounding surfaces of the temporal lobes and of the crura cerebri, likewise revealed marked injection of the vessels and the presence of a yellowish purulent exudate. Cultures of the exudate were made under careful aseptic precautions. The brain was then placed in fixing fluid for later study.

Cord: On removal of the cord and opening up of the dural cavity, the pia was injected and covered, in the cervical and lumbar regions, by a yellowish purulent exudate. Cultures were made from this exudate.

Brain: After the brain had been sufficiently hardened it was sectioned anteroposteriorly by direct cross section beginning at the anterior portions of the cerebral hemispheres and continuing, by parallel section, to the cerebellum. In the right centrum ovale and at the region of the first portion of the anterior horn of the right ventricle petechial hemorrhages were present in the roof and lateral wall of the ventricle. This petechial hemorrhagic condition was still present, especially in the head of the corpus striatum on the right side. The optic thalamus of the right side showed punctate hemorrhages. The ventricle, including the anterior and middle horns, was filled with a purulent exudate. There was apparently no invasion of the hemorrhagic infiltration into the lenticular nucleus or the internal capsule or into the claustrum on the right side. The right ventricle in the region of the posterior horn appeared to be free from purulent exudate. The left ventricle in the anterior portion of the middle horn and in the entire posterior horn showed purulent exudate. The basal ganglions on the left side were apparently free of petechial hemorrhagic areas. The purulent exudate was marked on the pia covering the basal ganglions, particularly in the recesses of the crura cerebri. The portion of the white matter and basal ganglions affected by the punctate hemorrhages showed undue softening.

Microscopic Findings: The most important and relevant changes were found in the brain and cord. The meninges of both brain and cord showed a purulent exudate. The bacillus isolated by culture, both antemortem and postmortem, was found present in this exudate both by Gram-Weigert and thionin stains:

"pari passu" we have used the thionin stain in cases requiring the demonstration of organisms in the tissue as a control on the Gram-Weigert stain, particularly in cases of gram-negative bacteria.

Sections showing the roof of the ventricle, including a portion of the centrum ovale, exhibited petechial hemorrhages and perivascular small round cell infiltration. The ependyma of this ventricle was infiltrated by pus cells among which the bacillus was found by Gram-Weigert and thionin stains. The sections of the head of the right corpus striatum showed distinct inflammation with petechial hemorrhages and perivascular infiltration. Sections of the pons and medulla were normal, except for the presence of the purulent exudate on the pia. Cross sections of the crura cerebri taken in the region

Fig. 2.—Brain, showing lesions grossly.

of the origin of the third nerve showed perivascular round cell infiltration but no petechial hemorrhages. Where the nerve emerged the pia showed considerable purulent exudate.

Sections of the subdural hemorrhagic tumor all showed an extension of the meningitic exudate into the bloodclot.

Cross sections of the cord at different levels showed a pure septic meningitis. The organism was again demonstrated by stain.

Bacteriologic Data: On March 30, 1919, one day before the death of the patient, a specimen of spinal fluid was received at the laboratory. It showed 12,000 cells of which 90 per cent. were polynuclears and 10 per cent. mononuclears, with globulin in excess. A stained smear showed the presence of a small gram-negative motile coccoid bacillus. The fluid was cultured, and

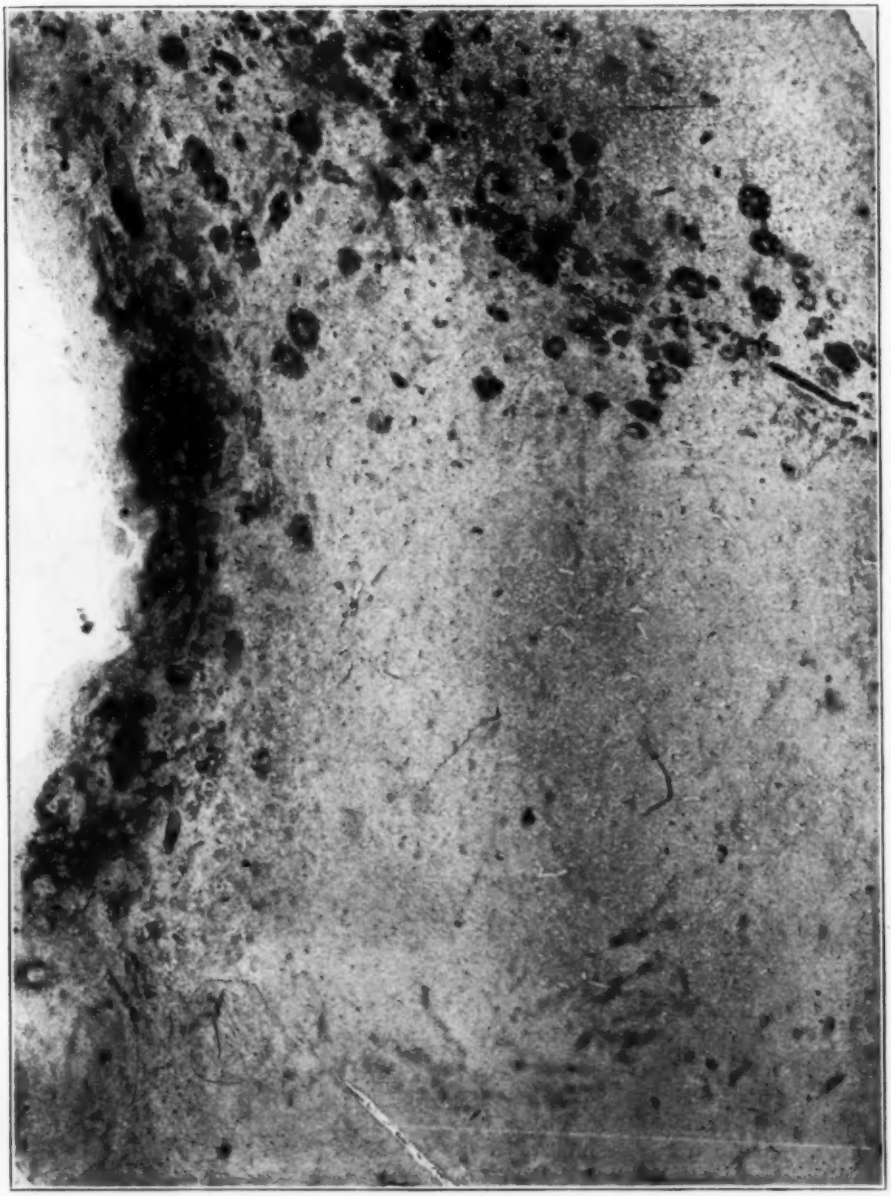

Fig. 3.—Section showing centrum ovale and ependyma of ventricle. Note petechial hemorrhages in the substance of the centrum ovale and acute purulent ependymitis.

a pure growth of this bacillus was obtained on both plain and blood-enriched mediums. In size it was 0.8 micron by 0.3 micron. In subsequent cultures the size varied greatly, some of the bacilli approaching 1.2 microns in length and some suggesting polar bodies.

Inoculations were made into a guinea-pig, a mouse and a rabbit. These animals, after a lapse of from about three to seven days, died. While sick none of them showed any stupor. Necropsy showed purulent peritonitis only, all inoculations having been intraperitoneal. The organism was recovered in pure culture from the abdominal cavity of each animal. The rest of the organs of the animals were normal, including brain and cord.

On April 1, 1919, at the time of necropsy on the body of J. K., cultures were made from the cerebral cortex and the purulent exudate on the base of the brain and the exudate on the cord. All of these cultures showed the same organism as that found in the antemortem cultures of the spinal fluid. An intravenous injection of 0.5 c.c. of bouillon culture was made into a rabbit. This injection was made April 7, the animal dying April 11. No evidence of stupor was observed while the animal was alive, although it appeared quite sick and disinclined to move about. Necropsy made on this rabbit revealed no gross lesions in the organs; and when we studied them, none of the animals inoculated showed any gross or microscopic evidence of infection of the brain or cord. The organism was recovered in pure culture from this rabbit's heart's blood.

Two guinea-pigs were injected intraperitoneally with cultures from the cerebral cortex: one lived seven days, the other three days. Necropsy showed purulent peritonitis. The organism was recovered from both animals in pure culture. We are still unable to classify the organism. We tested the original cultures obtained antemortem from the patient's spinal fluid, postmortem cultures and cultures from the inoculated animals. All grew on the chosen mediums, and all reacted alike on the sugars. No characteristic growth was observed on any of the mediums which would aid us in differentiating it from other organisms of like morphology. On sugars the following reactions were noted:

Glucose	Acid—Gas
Lactose	No change
Saccharose	No change
Mannite	Acid—no gas
Inulin	No change
Dextrin	No change
Maltose	No change
Galactose	Acid—no gas
Levulose	Acid—no gas
Litmus milk	No change

No indol production

Agglutination test with typhoid and paratyphoid A and B serums, negative.

The organism grew best aerobically. Its motility was quite marked in fresh cultures. In older cultures its motility was constantly lessened.

The cultural characteristics of this organism on sugars coincide with those of no other organism of which we know. Its action on the sugar mediums was tested several times and agreed with the original findings each time. There is no doubt that this organism was the cause of the patient's meningitis; and in

Fig. 4.—Section of corpus striatum; marked hemorrhagic extravasation, thrombotic vessels and exudative inflammation.

view of the fact that it was demonstrated in the purulent exudates of the ependyma of the ventricles, we are of the opinion that it probably caused the inflammatory changes and petechial hemorrhages of the centrum ovale, corpus striatum and the optic thalami.

SUMMARY

From the clinical standpoint our case was one of meningo-encephalitis, with lethargy and involvement of the motor fibers of the third, sixth, seventh, tenth and twelfth cranial nerves.

The etiologic cause was a gram-negative motile bacillus, unidentified, but probably belonging to some intermediate class of the colon-typhoid-enteritidis group.

Pathologically, the lesion demonstrated septic meningo-encephalitis and ependymitis, with punctate hemorrhages and perivascular cell infiltration of the centrum ovale, corpus striatum and optic thalamus.

We are indebted to Drs. Nidish and Atchley of the laboratory staff, and to Dr. Peter Lavelle of the intern staff, for their cooperation and the work which they did on this case and to suggestions and aid given by Dr. John H. Larkin of Strecker Memorial Laboratory, City Hospital. For permission to publish this case we wish to express our thanks to Drs. Hall and Merrill, on whose service the case occurred, and to Dr. C. F. Barber, president of the Medical Board of Kings County Hospital.

Abstracts from Current Literature

TRAITEMENT INTRARACHIDIEN DES AFFECTIONS SYPHILITIQUES ET PARASYPHILITIQUES DU SYSTEME NERVEUX. Gonzalo R. Lafore, Rev. neurol. **26**:625 (Aug.) 1919.

The author reviews the works of Nonne, Plaut and others, showing that general paralysis and tabes are of syphilitic origin; Noguchi and Moore's demonstration of the spirochete in the cerebral cortex of paretics and in the cord of tabetics; Noguchi's proof of spirochetes in parasyphilitics, and the work of Nichols, Houghman and others who demonstrated the existence of the spirochete in the spinal fluid of monkeys and other animals after infecting them through the testicles and the conjunctivae.

He then raises the question of why treatment has not been efficacious in the so-called parasyphilitics (paretics and tabetics). He reviews the discussion of Goldman, Weed, Frazer, Dickson, Halliburton and others on the subject of the origin and the movement of the spinal fluid. He does not accept the idea of Mott, Dickson and Halliburton that the fluid passes from the subarachnoid space through the capillaries, etc., as he thinks that the greater pressure is on the side of the blood stream, pressure in the veins being less than that of the cerebrospinal fluid. Toxic substances secreted in the brain by the spirochete pass into the perivascular lymph current; from the cranium they pass to the spinal canal.

There is, however, a current, he thinks, moving in the opposite direction (as has been demonstrated in the case of tetany treated by the method of Doyen). These two counter currents converge in the direction of the bulb.

By animal experimentation the author has produced through spinal injection a diffusion of coloring matter through the cord, bulb, base, encephalon and the olfactory nerve, as well as by penetration through the ependymal canal. If the animal lives long enough the convexity of the brain and the surfaces of the longitudinal fissures also show the coloring matter. Intraventricular injections show coloring matter in the aqueduct of Sylvius, the fourth ventricle, Ammon's horn and the like.

That mercury and arsphenamin have appeared to irritate the cortex is seen in speech difficulties, nausea, headaches, etc. He cites the case of Cotton's patient who had had an intraspinal injection one day, who died the next, and in whose ventricle, arsphenamin was found the following day. The author himself had a case in which he demonstrated that arsphenamin rose from the lumbar region as high as the cerebral region in half an hour.

The author agrees with Leredde on the importance of treating the infection rather than the symptoms and believes that treatments should be pushed as long as laboratory findings are positive, even though clinical manifestations have disappeared. He does not agree with Dejerine who maintains that the mercurial treatment of tabes and general paresis differentiates these from secondary syphilis, but he does agree with Leredde that vigorous intravenous and intraspinal injections bring about important changes in the cerebrospinal fluid in tabes and, though not so much so, in general paresis. He thus quotes Leredde's résumé:

1. The result of the treatment is certain and shows itself in all the changes of fluid.
2. Lessening of lymphocytosis is the rule, and may be quite marked.
3. Hypertension usually disappears.
4. In contrast to the results in general paresis, the findings of the fluid in tabetics yield very strikingly to neo-arsphenamin.
5. Quantitative diminution of albumin and globulin runs parallel with the disappearance of the serologic reaction.

Many of the author's patients who have not yielded to mercurial preparations and to intravenous treatment have improved remarkably under intraspinal treatment. Certain patients call for both intravenous and intraspinal treatment because of an involvement of the vessels, viscera and general body structures as well as of the central nervous system.

The ideal method in the treatment of syphilis of the central nervous system is that which reaches readily the site of the lesion in a direct way and in exact dosage and not in a manner that depends on the varying filtrating qualities of the choroid plexus of the brain or the cord.

The author uses the terms "benign" or "latent" for those cases of syphilis in which the patient never has known that he had syphilis and for those cases in which the patients have had a chancre which was considered nonsyphilitic and which disappeared following local treatment.

Laredde finds that 50 per cent. of his female cases and 28 per cent. of his male cases fall under this classification; 40 per cent. of the author's tabetics and 35 per cent. of his general paretics also fall under this classification.

The laboratory findings frequently show syphilis of the central nervous system before any clinical symptoms appear and so permit diagnosis of the early stages of general paresis which frequently is looked on as neurasthenia. It is these patients that are benefited most by treatment

The Diagnosis of General Paresis and Cerebrospinal Syphilis.—The author then enumerates the five laboratory signs and reactions for the differential diagnosis of general paresis and cerebrospinal syphilis: (1) Wassermann reaction on the blood; (2) increase of globulins; (3) lymphocytosis and presence of plasma cells; (4) Wassermann reaction on fluid, and (5) Lange's colloidal gold test.

The first three of these reactions may be present, either in paresis or in cerebrospinal syphilis but the fourth and fifth may not appear. The author states that from 80 to 95 per cent. of all patients show a positive Wassermann reaction on the spinal fluid if not more than 0.2 c.c. of fluid are used, but the reaction is usually negative in syphilis and tabes. Only 20 per cent. of tabetics show a positive Wassermann reaction on the fluid. Those patients with syphilis of the central nervous system in whom there is a negative fluid reaction may show a positive fluid reaction on using from 0.3 to 1 c.c. of fluid.

The author then raises the question whether it is possible, by the aid of the Lange reaction, always to differentiate general paresis from other forms of neurosyphilis. He thinks that one may be reasonably sure of the diagnosis of paresis if it has the usual clinical symptoms together with the five laboratory reactions, but he believes there are certain cases of the so-called pseudo-general paresis which do not show all the laboratory signs but present the clinical symptoms, and yet at necropsy and by histopathologic study fail to show microscopic changes characteristic of general paresis as established by Nissl and Alzheimer. He then quotes Southard and Salomon's belief that in doubtful cases the patient ought to have the benefit of modern treatment because there are cases in which it is impossible to determine whether the patients are paretics or other types of neurosyphilitics.

The author has seen patients in his clinics regarded as paretic without presenting any characteristic mental symptoms and running a course of ordinary neurosyphilis, and also patients with tabes with optic atrophy who have given laboratory reactions almost identical with those of general paresis. One of his patients had a gummatous lesion involving almost the entire hemisphere; another showed multiple gummas and yet the diagnosis could not be made until necropsy.

Suggestions for Treatment.—The author offers these suggestions for treatment:

1. In the treatment of cases of simple neurosyphilis as judged by the Lange curve, but with positive blood and negative fluid reaction and with an increase of globulins and cells, use may be made of the intravenous method and, if after four or five injections, improvement is not evident, alternate intravenous and intravenous and intraspinal treatment.

2. In the treatment of cases of simple neurosyphilis with both the blood and fluid reaction positive, one should begin with the combined intravenous and intraspinal treatment.

3. In the treatment of cases of simple neurosyphilis with negative blood and positive fluid one should begin with intravenous treatment; Wassermann tests should be taken on several bloods and if they continue negative only intraspinal treatment should be used. Almost all these cases have some concomitant vascular or visceral involvement, and the combined method of treatment is the surer.

4. In the treatment of parasyphilis (tabes and general paresis), both the intravenous and intraspinal method should be employed from the beginning.

The author reviews the various opinions on the relative value of intravenous and intraspinal treatment, especially in cases not tabetic or paretic, and the still more debatable question of the so-called cure of general paresis and tabes. He admits that there are a few indubitable cures by the intravenous method alone, but he emphasizes the fact that there are many more patients that have not been benefited by this method alone, but that quickly improve as soon as intraspinal treatment is introduced. He then cites instances of five such cases in his own practice, including a case with beginning optic atrophy and spastic paraparesis, a case of tabes, one of general paresis, one of vascular syphilis with pseudobulbar lesions, and a case of syphilitic meningo-encephalitis with paraplegia. His account shows rather remarkable improvement, but he does not state whether they are selected cases or not.

Preparation of Serum and Technic of Injection.—The author then describes the technic of his collaborator for the preparation of the serum in vitro. It is put in a flask with two openings, after the blood is drawn; the entire outfit is sterilized in an autoclave at 120 C. After the serum is prepared, it is sealed up in ampules containing 4 mg. of mercuric chlorid and 7 mg. of neoarsphenamin and employs such fractions of the contents as he wishes to use in individual cases. If made carefully, the ampules may be used for a period of several months.

The author's technic of intraspinal infection is:

1. Fifteen c.c. of fluid are drawn off for analysis.

2. The serum is mixed in 5 c.c. of the fluid and injected.

3. The author no longer makes preliminary use of stovain and procain as he has learned that their effects disappear before the onset of the pains due to injection. Instead he resorts to the routine use of pantopon and acetylsalicylic acid, three or four hours after the injection.

4. He prepares the serum according to the method of Byrnes and Ogilvie.

5. At the beginning of a course of treatment he uses 2 mg. of mercuric chlorid and 3 mg. of neoarsphenamin; later he uses as high as 4 mg. of mercuric chlorid and 7 mg. of neoarsphenamin.

6. Before beginning intraspinal treatment, he gives four or five intravenous treatments at intervals of from two to six days, using 1 cg. of mercuric cyanid and 0.3 gm. of neoarsphenamin.

7. He always uses in the first intraspinal infections arsphenaminized serum, never more than 2 to 3 mg. He has had no severe reactions.

8. In the interval (from twenty to forty days) between intraspinal injections, intravenous injections are employed but never more than two a week, and he always alternates the mercurialized and arsphenaminized serum.

Dosage Employed in Intraspinal Treatment. — Originally, the author gave large dosages producing strong reactions, maintaining such dosages up to the point of complete remission. Recently, however, he has begun his treatment with small dosages, never using enough to produce intense reaction, gaging the interval by the degree of amelioration of symptoms. Tabetics with marked crisis, paretics with seizures, and parasyphilitics with spasticity, should not receive over 2 mg. of mercuric chlorid and 4 mg. of neoarsphenamin.

At the end of six months at least, the patient should rest entirely for from two to six weeks without treatment, in order to allow elimination of mercurial and arsenical products.

Effects of Intraspinal Treatment.—Besides the ordinary reaction familiar to us all, the writer speaks of:

1. The Subjective Order of Curative Effects. The symptoms disappear as follows: Lancinating pains, root pains, headaches, paresthesias, pareses, contractures and general mental symptoms.

2. Objective Order of Curative Effects: There is improvement in everything that does not remain as a result of an irreparable sclerosis, such as, impaired pupillary reactions, optic atrophy and absent knee jerks. The more recent and more active the process, the more likely the recovery. The author has seen a reappearance of knee jerks and pupillary reflexes. In general paretics, the Wassermann reaction on the fluid is the last to disappear; the Lange curve changes less quickly than the Nonne and Noguchi reactions. When the curve does change, it first becomes similar to that of cerebrospinal syphilis and finally it becomes normal.

Laboratory changes are always slower than the clinical, the latter frequently clearing up in from three to four months. The laboratory changes, therefore, serve as a guide for further treatment even after clinical symptoms disappear; patients with only slightly advanced cases of paresis usually require a year and a half of treatment, although they may show complete remission in nine or ten months; tabetics usually require from seven to eight months; advanced paretics of more than a year's standing may show temporary improvement but progressively get worse in spite of intensive treatment.

Because certain critics maintain that the alleged cures are nothing more than a coincidence of spontaneous remission accompanying treatment, the author feels obliged to discuss the question of so-called spontaneous remission.

Many paretics show spontaneous remission usually following variable periods of pseudoneurasthenia, followed by a period of excitement with delusions of grandeur or persecution. If treatment is resorted to before the spontaneous remission, it is easy to argue that one is not safe in ascribing the remission to the treatment, but the fact remains that many paretics do not show spontaneous remission. The writer quotes the percentages of Cotton and of Southard and Salomon which show certain advantages of treatment in spite of the fact that they reported nonselected cases. He is willing to admit that cases of more than a year's duration do not give a good prognosis, although the patients may be helped; but he does insist on the fact that patients found early, show complete remission of both clinical and laboratory symptoms, and this is the distinguishing feature between spontaneous remission and that due to treatment. In spontaneous remissions, the reactions on the blood and the spinal fluid usually remain positive, and if they show any change, this is very slight.

Real and Apparent Aggravation of Symptoms in Treated Cases.—General paretics ordinarily show a certain amount of mental confusion and increased difficulties in speech and in writing; tabetics show difficulty in walking and ataxia, but this usually disappears in from eight to ten days following injection. There are a few cases, however, in which aggravation of earlier symptoms is persistent. Sometimes certain infectious diseases, such as influenza, have a similar effect. The author had one tabetic whose symptoms were aggravated transiently just after vaccination.

Permanency of Results.—Nothing can yet be affirmed as intraspinal and intravenous methods have been in use only a few years. The author cites the case of a patient with paresis, first seen in December, 1916. All clinical and laboratory signs had disappeared by November, 1917, and had not reappeared when the patient was last seen in February, 1919, without any other treatment than an occasional intravenous and intraspinal treatment every three or four months.

The author then submits the history of treatment and results in the case of five general paretics, noting results as follows: One recovery; one improved but with very slow and mild progression; one almost recovered with great amelioration; one with unusual rapid clinical improvement; and one with rapid clinical improvement and modification of laboratory signs.

The conclusions which he reaches are based on the study of fifteen cases of general paralysis, eight of tabes, four of syphilitic spinalparaplegics, and two of syphilitic cerebrospinal syphilis. He omits his advanced cases of general paralysis and such other patients as were under his care for only a short period.

Conclusions.—1. The indication for exact intraspinal treatment is furnished by positive Wassermann reaction on the fluid and low Lange's colloidal gold reaction.

2. The efficacy of such treatment is strikingly manifested in those syphilitic processes which have not as yet involved the deeper portions (parenchyma) of the nervous system.

3. In general paralysis, such treatments do not give results if cases are of more than six or eight months' standing.

4. In tabes, results are almost always good; subjective symptoms disappear rapidly and a large number of the objective, likewise.

5. In syphilitic optic atrophy, intraspinal treatment is most favorable for arresting a rapidly progressive blindness.

6. In cases with radicular involvement, pains disappear much more quickly than under intravenous treatment.

7. In combined system diseases, especially sclerotics, treatment is of little value unless the cases are caught early.

8. Remissions which occur in cases of general paralysis may persist for years.

9. It is too early to maintain, because of the few years in which intraspinal treatment has been employed, that we may bring about definite cures in general paralysis although we may affirm that this may be the case.

10. Remissions due to intraspinal treatment differentiate themselves from spontaneous remissions in that the latter show only temporary amelioration of clinical symptoms, and laboratory symptoms still persist. In contrast to these, remissions due to therapeutic measures show true cures with amelioration of both clinical and laboratory symptoms but the touchstone of treatment in general paralysis lies in diagnostic skill which one may fortify by the aid of the five laboratory reactions.

12. The author's method, if followed with rigorous precautions, is harmless. These precautions include careful diagnosis of the case, and mild therapeutic measures, intravenous and intramuscular, to determine the patient's resistance to antisyphilitic remedies.

JONES, Detroit.

STUDIES ON EPINEPHRIN. I. EFFECTS OF THE INJECTION OF EPINEPHRIN IN SOLDIERS WITH "IRRITABLE HEART." J. T. WEARN and C. C. STURGIS, Arch. Int. Med. **24**:247 (September) 1919.

The authors report their results with the Goetsch test in cases of "irritable heart" of soldiers. Goetsch has reported that cases of hyperthyroidism give a very marked reaction to small doses of this drug. While a large number of these cases of "irritable heart" gave a positive reaction to the Goetsch test, studies on the basal metabolism and a careful clinical analysis of the cases do not lend support to the theory of hyperthyroidism as a causal factor in "irritable heart." The technic employed by Wearn and Sturgis in carrying out this test was as follows: The patients were put to bed at absolute rest for one hour, and at the end of this period control readings of the blood pressure, pulse and respiratory rates were made at five minute intervals. At this time note was also made as to the presence of nervousness, precordial pain, palpitation, dizziness or any other symptoms; likewise, the objective condition of the patient was observed, and the presence of any of the following signs was recorded: tremor of the hands, sweating, coldness of the hands, throbbing of the neck, epigastrium or the precordium, and pallor or flushing. At the end of the hour the patient was usually quiet, and the blood pressure and heart rate were normal (in contrast to the patient with exophthalmic goiter in whom a marked tachycardia persists even when at rest in bed). After satisfactory control readings were obtained, 0.5 c.c. of a 1:1,000 solution of epinephrin freshly prepared was injected deep into the deltoid muscle. The onset of the reaction occurs about twelve minutes after the injection, the height is reached on an average in thirty-two minutes, and the reaction finished in approximately one hour. A positive reaction is characterized by a rise in blood pressure of 10 mm. or more, associated with a rise in pulse rate of at least 10 to the minute (provided that these are not isolated rises), and is associated with tremor, pulsation, nervousness, precordial pain, dizziness and restlessness. Cases with a fairly marked rise in blood pressure and pulse rate without other changes were also regarded as giving positive reactions.

Twenty-six normal soldiers were used as a control, and in not a single case was there a positive reaction to epinephrin, as judged by the rule formulated to determine a positive or negative case. The blood pressure and pulse curves showed slight, if any, change, and the characteristic subjective and objective conditions of the epinephrin sensitive group were lacking. In distinct contrast to the epinephrin sensitive patients, with marked restlessness and anxiety, the controls remained perfectly quiet, some of the men reading throughout the greater part of the test, while others actually slept.

Of seventy-three cases of the group of soldiers with "irritable heart" whose symptoms were of long standing, Wearn and Sturgis found that forty-three, or 59 per cent., showed a definite hypersensitiveness to epinephrin or gave a positive reaction as described above. Twenty-seven, or 37 per cent., showed no reaction. Three cases, or 4 per cent., gave a doubtful reaction. Of eighteen cases of "irritable heart" in which the symptoms dated to some recent infection, such as rheumatic fever or pneumonia, six gave positive reactions to epinephrin and twelve gave no reaction. Observations made on a group of cases in which the symptoms developed following some nervous or physical strain, showed, that of seven of this type, five gave a positive reaction and two gave no reaction. The proportion of positive reactions in cases of "irritable heart" is in distinct contrast to the complete absence of positive reactions among normal, trained soldiers.

The authors suggest that the symptoms and signs induced in the positive cases are the result of a hypersensitive, sympathetic, nervous system. This belief is based on the fact that epinephrin has a selective action on the sympathetic nervous system. They state that a careful clinical analysis of the patients showing positive and negative reactions revealed no essential clinical differences between the two groups. They are unable to state that the clinical condition of the patients who are sensitive to epinephrin depends in any way directly on the factors underlying the positive reaction, or that the factors causing the symptoms in the positive and negative groups are the same. Although all efforts were made to improve the physical condition of these men, not one of those who gave a positive reaction, and only three who gave a negative reaction, were able to do full duty. The authors conclude that this class of patients is not a true product of the war, and that they are seen not only in military but also in civilian life. They are the type that go from one outpatient clinic to another with complaints of nervousness, weakness, palpitation, precordial pain and dyspnea; they are classed as neurotic or neurasthenic individuals and have usually been receiving medical attention for years. There seems to be a certain level of strain below which these people can live without symptoms, and their one effort in life is to keep below this level, above it their symptoms appear and they break.

STUDIES ON EPINEPHRIN. II. THE EFFECTS OF EPINEPHRIN ON THE BASAL METABOLISM IN SOLDIERS WITH "IRRITABLE HEART," IN HYPERTHYROIDISM AND IN NORMAL MEN. E. H. Tompkins, C. C. Sturgis and J. T. Wearn, Arch. Int. Med. **24**:269 (September) 1919.

Four groups of subjects were studied. Two of these groups were negative to epinephrin and showed no reaction, while two were positive to epinephrin and responded with the typical reaction of the hypersensitive subject. The groups positive to epinephrin consisted of patients with "effort syndrome"

("irritable heart") who reacted to epinephrin and a small series of cases of hyperthyroidism. The groups negative to epinephrin consisted of normal subjects and patients with "effort syndrome" who did not react to epinephrin.

With unbroken regularity the gaseous metabolism showed a rise after the injection of epinephrin (0.5 c.c. of 1:1,000 epinephrin solution). This is true in the normal, as well as in the pathologic cases, and whether there is any other sign of reaction or not. This increase in metabolism, however, is far more marked in cases with a positive than in those with a negative reaction, and it is parallel to the pulse, blood pressure and symptomatic changes. With one exception, every positive reaction was accompanied by a rise in metabolism of 20 per cent. or over. On the other hand, no negative reaction showed a rise above 20 per cent. and in the majority of cases not above 15 per cent.

The increase in the metabolism is expressed partly by a greater oxygen deficit in the expired air, but far more by an increased amount of air breathed, as is shown by the greatly augmented minute volumes. The increase in the minute volume is as invariable as is the rise in metabolism. The amount of air breathed per minute was increased as much as two liters in a considerable number of cases. There is an increase of the respiratory rate or depth, or both.

There is frequently an elevation of the respiratory quotient. The maximum of this elevation normally precedes that of the metabolic increase. The increase in metabolism is not due to excitement attendant on injection or the stimulation of heart and respiratory muscles resulting from injection. (The basal metabolism in cases of "irritable heart" with increased respiratory activity shows no changes, and after injection of atropin, although the pulse rate is increased, no metabolic increase occurs.)

The authors conclude that epinephrin acts indirectly in causing an increase in the metabolism, and it is by acting through the sympathetic system that it produces the changes—increased muscular tonus, tremor and hyperglycemia—which are probably the actual causes of the increased metabolism.

Finally they conclude that as epinephrin is a stimulant to the sympathetic system, the differences in reaction and metabolic response in the negative and positive cases are probably due to differences in that system.

MYERS, New York.

CONTRIBUTION TO THE STUDY OF AUTOMATISM IN THE MEDULLA. ABRAMO LITVAK, Riv. di patol. nerv. **24**:15 (March 25) 1919.

The author refers to Luciani's law that the excitement of centripetal nerves may awaken flashes of consciousness and motor impulses, preceded by the choice of centrifugal pathways through which the stimulus is transmitted, and also to Wundt's conception that tendon and superficial reflexes are voluntary movements become automatic by filogenetic development. The finding of nerve terminals in tendons disposed of Westphals conception that tendon reflexes were due to direct stimuli applied to muscles through their tendons. With these preliminary remarks, the author introduces a new abnormal plantar reflex. His discovery disproves the contention of Marinesco that in paralysis of the lower extremities the extensor center alone remains functionating because in his cases (Figs. 2 and 3) both the extensor and flexor abnormal plantar reflex were demonstrated. While he has noted the abnormal flexion of the toes caused by stimuli applied to the tendo achilles, thigh, abdomen, etc., in cases of hemiplegia due to capsular or cortical lesions, his sign has been observed only in lesions of the peduncles above the fibers for the arm. This is an

important localizing sign. However, the toes occasionally assumed the extensor Babinski type and the reflex was not always in flexion. In many normal persons and in hemiplegics with lesions in other sites, grasping the paretic hand never caused either the author's sign or the Babinski phenomenon.

Case 1 illustrates a vascular accident to the left side of the pons; therefore all the abnormal signs must originate in the medulla. The "sincinesie" and associated movements in these cases depend on the intensity of the stimulus, the condition of the centers receiving it, the length of the pathway traversed and the resistance offered by it. Babinski long ago noted that "sincinetic" movements were more readily brought out by stimulating the paretic limb. The author noted a diffusion of these associated movements from the big toe to the feet, leg and thigh, and while these movements always spread from the upper to the lower (from bulb to cord) extremity, the contrary is never true. This bears out Sherrington's law of caudal radiation of spinal reflexes in mammals. In the author's case, it was always possible to provoke movements in the lower extremity by strongly applied or painful stimuli to the upper extremities, but never if the stimuli were applied to the lower extremities. In this case both the flexor (author's sign) and extensor responses of the toes were obtained. The flexion of the toes of the paretic foot was obtained either by (1) reflex stimuli applied to the other thigh or (2) strong pressure applied to either the sound or paretic hand. The extension of the toes of the paretic foot (right) was obtained by stroking the plantar surface (Babinski sign) of the paretic foot, or by applying the stimulus to the thigh of the paretic leg (Oppenheim).

The author's patient also had a paralysis of the left sixth cranial nerve and a complete hemianesthesia to all types of sensibility; also a bilateral clonus and absent left plantar reflex. All abdominals were absent.

By applying a mild stimulus, extension of the toes (Oppenheim) was obtained; by pricking more strongly the paretic thigh, flexion of the toes was obtained, even if this was done to the arm or abdomen of the same side. When the left (sound) leg was pricked, the right paretic leg was always withdrawn.

The author believes that the flexion reflex of the toes of the paretic side was pathologic and like all defense reflexes had its center in the medulla because: 1. There was always greater response in flexion or in extension when the plantar surface of the paretic leg was stroked corresponding directly to the strength of stimulus than was obtained by the same stimulus in the other leg. 2. There was no paralysis of the left leg. 3. All the abnormal associated movements were very active on the right and hardly elicitable on the left side. 4. Flexion of the toes on the right was obtained by stimulating other regions of the right extremities. This flexion reflex in the toes, like the retraction in flexion of the paretic leg, is a medullary reflex function. Because this reflex is objectively like the normal, it might be difficult to determine in a given case by this alone which is the paretic side.

In the author's second case, attention is drawn to Dagini's two signs; the oculocardiac reflex and the adduction of the paretic arm on pressure of the eyeball.

The author's third case illustrates his point that the abnormal associated movements seen in paretic limbs are present in direct proportion to the degree of paralysis, that is, the condition of the center receiving the stimulus. For that reason neither the abnormal extensor Babinski or flexor response was obtainable in the right paretic leg because this was little involved. The normal plantar reflex was absent, but when the eyeball was pressed, the pulse

slowed up and the paretic arm was adducted showing that the automatic action of the medulla was free from cerebral inhibition through the corticobulbar and corticospinal tracts.

The next part of the author's extensive article is devoted to discussion as to what influence mild pyramidal lesions, as shown by the Babinski sign, and severe lesions, as shown by the Marie Foix sign, have on the automatic functions of the medulla.

The Babinski phenomenon is elicited not only by plantar stroking but also by stimulating various antagonistic groups of muscles of the paretic leg and even of the sound leg. Babinski has always contended that this phenomenon could coexist with a normal plantar flexion of the toes. Eight cases are briefly cited to show the variations in plantar reflexes in various types of hemiplegia and paraplegia, varying from complete absence to the Babinski phenomenon with flexion of leg and thigh. When the normal plantar reflex mechanism is absent independent centers in the cord react to cutaneous, osseous, joint and tendon stimuli, and responses are obtained from the toes even when the stimulus is removed to the leg, thigh and even the upper extremity. The classical, most frequent response is extension of the big toe, but at other times there is abduction of the toes or fanning. According to the author, the Babinski reflex, therefore, is not a plantar reflex but an expression of an abnormal reaction to stimuli, extending over a large zone. If this is true, then the flexion of the big toe provoked by stroking the thigh and the abdomen and by the author's method of squeezing the paralyzed hand, stretching the achillis tendon, etc., are not at all plantar reflexes but so-called defense reactions, improperly so designated, which have their center in the cord, bulb or subcortex, and not in the cortex which is the site of the mechanism of the normal plantar reflex.

The author believes, with Goldflam, that the center for the Babinski reflex is in the lumbosacral cord, and does not operate until the corticospinal inhibitions are removed. It has been noted in complete transverse lesions of the cord above the lumbosacral level, never when this is crushed. The fact, that the Babinski reflex is the normal response in the first year of life, is proof that it is a spinal function. In infants the reflex zone for this reflex is also wide, as it is also for the abdominals and the cremasterics. The author's twelfth case strengthens his belief that the Babinski is a spinal reflex. This patient had a tetraplegia with complete loss of all types, of sensibility and bilateral Babinski reflex. At necropsy, fracture of the second and third cervical vertebra with complete severance of the cervical cord was found.

Retraction of the leg (Marie Foix sign) is caused only by strong painful stimuli which contract the sartorius, adductors of the thigh and the anterior tibial group, or occurs in very profoundly paralyzed limbs. The author agrees with Marinesco that the Babinski phenomenon is the first to appear and the last to disappear. When the patient is dying or his medulla has become almost completely removed from the cerebral control by profound paralysis, stimuli produce a more widespread reaction of medullary and cord centers resulting not only in extension of the big toe, but also in retraction of the limb.

The last chapter deals with the nature of reflexes seen in moribund hemiplegics and paraplegics.

The sign of Marie Foix (described in 1853 by Sherrington in the spinal frog) is not so commonly seen as the Babinski, and is most commonly seen in the profoundly paralyzed. It occurs usually as the Babinski reflex disappears, and persists until death. Occasionally the Babinski reflex is seen in

the nonparetic limb and the Marie Foix sign on the paralyzed side; or a mild stimulus applied to the paretic leg produces a Babinski reflex, a stronger one causing the latter. Observations in many cases convince him that the Babinski reflex is a sign of milder pyramidal tract disturbance than the Marie Foix sign. In the presence of cortical and meningeal lesions other than the pyramidal tract injury this sign, however, loses its prognostic value. As an illustration, the author quotes a case of delirium with right hemiparesis, Babinski reflex on the right, Marie Foix sign on the nonparetic, left side. When delirium disappeared, the Marie Foix sign disappeared. The case was one of status epilepticus attacks with delirium at intervals of a few months, following a frontal skull injury which left him with a right hemiparesis. Another sign of profound paralysis, usually with a fatal ending, is the Dagnini "trigeminal-brachial" sign of adduction of the paretic forearm alone or with flexion of the forearm and hand on the arm. Dagnini's oculocardiac reflex is frequent in the young, but never seen in the aged. In many paralyzed patients the independence of the medulla from cerebral control favors the exhibition of retardation of this reflex. The author's Cases 13 to 19 illustrate these points. They are accompanied by graphic tracings showing the retardation of the pulse in the application of the oculocardiac reflex. In many of the patients showing the Dagnini and Marie Foix signs, there was also Cheyne-Stokes breathing which he also classes as an "ultimum moriens" reflex. The author in his discussion of these cases mentions another sign of Dagnini's, that is, the "frontalis" sign. The author declares that in supra bulbar pyramidal lesions the upper branch of the facial nerve also is involved with smoothing of the forehead on the paralyzed side (Case 19).

OSNATO, New York.

ETUDE SUR L'ORIGINE ET LA NATURE DE LA SCLEROSE EN PLAQUES. G. MARINESCO, Rev. neurol. **26**:481 (June) 1919.

There are two leading conceptions of the etiology and the pathology of multiple sclerosis: First, that it is inflammatory in origin, or vascular; second, that the inflammatory process is due to an infectious agent. It is not so much a sclerosis of nerve tissue or centers as it is a disseminated inflammation. It is especially in the acute and the subacute forms that the inflammatory character of the disease is best seen. The theory that the vascular lesions are secondary to those in the nervous tissue can no longer be accepted; on the contrary, the reverse is true, in spite of the earlier teachings of Charcot, Bourneville, Wernicke and others.

It is the opinion of Pierre Marie (and this is now accepted by Wernicke, Marburg and others) that the vascular lesion is in the form of a disseminated arteritis, the sequela of some well advanced primary condition, such as scarlet fever, pneumonia, erysipelas and similar diseases. Sometimes the arteritis appears toward the close of the conditions, or during convalescence, or even when recovery seems to be absolutely complete.

The lesions localize themselves by preference in the white substance, and follow the course of the smaller vessels, especially the veins. This explains the relative integrity of the nerve cells, but even these may be seriously damaged in a rapidly advancing case. The essential point to remember is that there occurs an infiltration, in and about the adventitia and the perivascular tissues, of lymphocytes, plasma cells, polyblasts and occasionally mast cells. Polymorphonuclears are the exception, and eosinophils do not occur. The proportions of these infiltrative elements vary with the stage of the lesion. In

the chronic or slowly advancing processes, lymphocytes and plasma cells predominate and should not be confused with the inflammatory type showing macrophages in the vessel walls. The chronic type shows changes not unlike those in general paresis; the inflammatory type follows only the disintegration of myelin into fatty bodies. Simultaneous with the infiltration of the vessel walls is the growth of the neurogliar cells. Giant neurogliar cells are found particularly in the cerebral cortex and in the optic nerve; and possess a marked capacity for fibrillogenesis.

The perivascular infiltrations are frequently nodular and not unlike lesions in syphilis, especially in general paresis. The adventitia does not constitute a barrier to the inflammatory process, as is evident from the invasion of lymphocytes, plasma cells, and polyblasts into the surrounding tissue.

The writer had two cases which showed inflammatory processes in the following important regions: periventricular, periependymal, and about the aqueduct of sylvius. More rare is an involvement at the level of the fourth ventricle or in the ependyma of the cord. This statement is in agreement with the opinion of other writers cited, and might lead to the conclusion that the spinal fluid is the agent through which infection reaches the areas just mentioned, and that the virus gets to the fluid through the medium of the lymph paths. Consequently, wherever the vessels and their corresponding lymphatics are involved, there will be a lesion of nervous tissue. This explains the frequent involvement of the optic nerve in multiple sclerosis.

Marinesco's studies do not lead him to accept Ribbert's conclusion that vascular thrombi occur as the result of inflammatory processes in multiple sclerosis. The intima of the vessels, like the gray matter of the brain and cord, appears to escape. The virus in the lymphatic circulation affects chiefly the nutrition of the myelin, but also may bring about a secondary degeneration of the axis cylinder, although even in this case there is usually collateral regeneration.

The remissions and exacerbations of multiple sclerosis are more in harmony with the theory of an infectious process than a toxic (due to lead, mercury or other minerals). While fever and chills in certain cases have led certain authors to look on multiple sclerosis as due to influenza and other similar diseases, it is more likely that it is a specific infectious disorder, and while similar in certain respects to syphilis (particularly general paralysis), yet it is evidently not due to the spirochete of syphilis. Although Nonne in a study of seventeen cases of multiple sclerosis obtained a positive Wassermann reaction in three cases, the fact remains that neither clinical nor histologic examination points to syphilis in multiple sclerosis. Moreover, certain writers, like Collins, who give a routine Wassermann test in all their cases of multiple sclerosis, report all reactions negative.

It was not until September, 1917, that Kuhn and Steiner, by injecting the blood and spinal fluid of patients affected with multiple sclerosis into guinea-pigs and rabbits, demonstrated a spirochete in rabbits. They injected blood, spinal fluid and a mixture of both into the peritoneal cavity, the heart and the cavity of the eyeball of these animals, and obtained, in from three days to two weeks, disturbances of motility, especially a staggering gait, more pronounced in the hind limbs. This condition lasted from a few hours to nine days, and in the fatal cases was followed by paralysis and death. Examination of the blood drawn from the eye or ear of the animal revealed the presence of spirochetes which, in form and size, resembled those that produce hemorrhagic icterus. They were spiral in form and often showed a refracting point

at the extremity. They were also variable in form, and either extremity might be provided with a hair-like prolongation. In the multiple sclerosis of man, they have not seen the spirochete.

By subdural injections, Simon has demonstrated the transmissibility of the virus in rabbits and has observed paralytic manifestations followed by death. In March, 1918, Siemerling announced that by the aid of the ultramicroscope, he had found spirochetes in the brain of a patient that had had multiple sclerosis. They presented the same characteristics as those described by Kuhn and Steiner.

In October, 1918, Marinesco began experimental work with two patients who showed many of the clinical symptoms of classic multiple sclerosis. He made intracerebral, intraperitoneal and intraspinal injections of the spinal fluid of these patients into six guinea-pigs. Two of the pigs showed, three or four days after the injection, motor disturbances in the posterior portion of the body, especially in the form of disturbed gait. Spinal fluid was then withdrawn from the fourth ventricle of these animals and a considerable number of spirochetes, like those described by Kuhn and Steiner, were seen under the microscope. M. Pettit and Marinesco himself, in certain later attempts at transmitting the virus, obtained negative results. This proves only that the parasite is not always in the spinal fluid, or if it is there, it is present only in reduced numbers. Moreover, the two patients from whom the inoculating fluid was drawn were in a more severe and advanced stage of the disease; as a consequence, the parasites were probably less numerous and virile.

Summary.—While the research is still incomplete, it brings to light two new facts: 1. Multiple sclerosis is transmissible due to the virus of a living organism. 2. This virus is the product of a spirochete entirely distinct from the *Spirochaeta pallida.* These facts on first consideration do not seem to harmonize with the isolated character of the disease which is neither endemic or epidemic, nor is it contagious; and it appears to confine itself to the nervous system.

Reviewing all the facts, however, multiple sclerosis appears much more like an exogenous malady of infectious nature, than like an endogenous malady as maintained by Strumpell. The course of the disease, the successive exacerbations, and the appearance of the fundus permit us to differentiate, in the living subject, the various phases of the disease (really stages of development of the parasite) which are not unlike those of syphilis, and lead us to conclude that the spirochete observed by Kuhn and Steiner, and by Marinesco is the pathologic agent of the disease. Multiple sclerosis, then, presents a series of well defined symptoms, and though varying in their clinical aspects, is a true nosologic entity and not a syndrome of sundry causes.

JONES, Detroit.

SCHIZOPHRENIA AND CHOROID PLEXUS. C. VON MONAKOW and S. KITABAYASHI, Schweiz. Arch. f. Neurol. u. Psychiat. **4**:363, 1919.

The writers call our attention to the fact that, until recently, we have attributed the normal and abnormal manifestations of the mind to a function of the cortex alone, and have looked on the secretory organs much as we have on the circulatory apparatus, that is, as a secondary source of assistance. It would be more logical to attribute our psychic life, consciousness and affect to a more closely associated function of these two structures. This view has phylogenetic and autogenetic support. In studying serial sections of a four

weeks old human embryo, we find that those portions of the dorsal alar laminae which subsequently become differentiated to form the choroid plexus, particularly in the vicinity of the vagus and glossopharyngeal centers, is always well developed; in like manner we find this structure developing in the median portion of the hemispheres. All of this proceeds with great rapidity, long before the essentially nervous elements of the medullary structure reach a corresponding degree of differentiation.

The writers look on this as a sort of overture for the later physiologic interchange between the choroid plexus on the one hand, and the rest of the brain on the other. In other words, the embryonic relation itself suggests the important rôle of the choroid plexus. The other glands of internal secretion also, particularly the suprarenals have, at the corresponding period of evolution, reached a surprising degree of differentiation. In short, the endocrine system is well developed at a period of fetal life when the essential structures of the brain are still lying in deep slumber. We can look on the organs of internal secretion as the mother source of our affects and instincts. In the grown up person, the choroid plexus in the fourth ventricle, as well as in the lateral ventricles, appears to be correspondingly less well developed than in the fetus; in lieu of this, however, they are much more intricately folded and villose. It has been demonstrated that the choroid plexus is a true gland, which not only secretes cerebrospinal fluid, but also neutralizes toxins derived from other glands and circulated in the blood. According to the writers, the substances so noxious to the brain are particularly epinephrin, thyroglobulin, and the secretions from the hypophysis, the epiphysis and the gonads.

The morphologic basis of the psychoses, particularly schizophrenia, has in times past been looked for and partly found in the cortex, while the internal secretory glands, particularly the choroid plexus, have been disregarded. The sources of our affect-life must be sought for primarily, not so much in the field of brain mechanics, as in that of biochemistry; those tissues of the brain must be subjected to the greatest morphologic changes which are most intimately related to the finer processes of nutrition and to the chemical changes that afford protection against substances circulating in the blood. These consist of the tela choroidea, plexus choroideus, the ependyma and the protective glia covering. The writers accordingly look on the choroid plexus, in the typical forms of schizophrenia, as not only functionally but also morphologically drawn into the circle of pathology.

Up to date, seventeen pathologic brains as well as a larger number of normal brains, have been studied. The former comprised not only cases of schizophrenia, but also other types of mental disturbances, such as progressive paralysis, tumors, etc. The authors were greatly surprised to find that the choroid plexuses of twelve patients, who had for years suffered from the more severe schizophrenic symptoms, were not found normal in a single instance. The results can be summarized somewhat as follows: hyperemia, diffuse degeneration of the villi, amyloid degeneration of the connective tissue, accumulations of colloid masses within the plexus and interpapillary exudation.

In other psychoses, not schizophrenic clinically, somewhat similar changes were found, but these were of much less intensity. Hyaline degeneration and a moderate atrophy of the choroid villi were observed in a few senile individuals and in chronic alcoholics. In a number of cases of schizophrenia, there were marked hyperemia to the point of capillary blood extravasations, thrombosis, and increase in the peri-capillary connective tissue with hyaline degen-

eration to the extent of complete obliteration of the villi. The pathologic process usually seems to begin with a cell infiltration in the tissue between the capillary walls and the secretory cells.

In view of the changes noted in the brains of schizophrenic patients investigated up to date, there remains little possibility, in contradistinction to other psychoses, that recovery without permanent defect may take place. The most serious consequences of a primary or even a secondary disease of the choroid plexus, the writers view in relation to the physiologic processes, since the innate protective power and resistance of the choroid plexus to substances harmful to the brain is lowered, and probably permits greater filtration, resulting in chemical combinations with the brain tissue itself, through the medium of the cerebrospinal fluid, against which the protective membrane of glia tissue is inadequate. Through disease of the plexus by way of intoxication, a disease of the cortex is eventually added; this would open a way for the development of typical schizophrenic phenomena.

In conclusion, the writers state their belief that this view would bear out the principle of minute division of labor, with the nervous system on the one side, and the glandular system on the other; and that a united action of the biophysic functions of the cortex and the biochemic functions of the glandular system would result in creating the highest phenomena of our psychic life.

WOLTMAN, Rochester, Minn.

MANIFESTATIONS NERVEUSES DU TYPHUS EXANTHEMATIQUE: 1. Reactions Meningees Tardives, II. Inegalite Pupillaire, III. Nevrites et Polynevrites. DEMETRE E. PAULIAN, Rev. neurol. **26**:650 (Aug.) 1919.

The author discusses the subject under these heads: I. Late Meningeal Reaction of Exanthematous Typhus.

In an earlier study the author showed, in collaboration with Devaux and Tupa, that there is always a marked meningeal reaction in the first days of the infection. He has also been able to establish an almost constant cytologic formula for this disease at its crisis and during the period of convalescence.

During the first few days of the fever, the spinal fluid on puncture shows an increase of pressure, and drainage has a salutary effect on the patient.

In a few grave cases with marked agitation and prolonged confusion, the spinal fluid is xanthochromatic.

All the fluids examined show a large number of cells, but no bacteria or parasites.

The cell changes are:

1. From the second to the fourth day there are lymphocytes, round cells, easily stained by basic dyes, the nucleus filling the entire cell body.

2. On the fifth day (when the patches appear) there are large mononuclears which persist during the period of eruption. The nuclei of these mononuclears may be eccentric, or only slightly so. There may be a few polymorphonuclears. They increase as the disease progresses.

3. About the twelfth day the mononuclears are replaced by lymphocytes, at the beginning easily stained and persisting well into convalescence, especially in cases in which the nervous system is involved; in some cases with nervous complications this condition may persist for even a year and a half.

Without the aid of the Wassermann test it would be difficult to distinguish some of these postexanthematous cases from syphilitic cases. In the typhus

cases there is always a high albumin content (as much as 0.50 or 0.65). Gentle heating in a test tube will produce a slight opalescence or milky whiteness.

A late lymphocytosis in the fluid is an important diagnostic feature and almost pathognomonic in individuals with nervous complications and especially in postexanthematous psychoses in which the spinal fluid (even in high quantitative tests) proves negative.

II. Cases in which There Are Inequality of the Pupils.

About 60 per cent. of patients show unequal pupils. If these patients are seen a considerable period after the exanthem has disappeared, one may mistake them for syphilitics. In the exanthematous cases the fluid, however, is always negative. The cytologic phenomena just described help clear the diagnosis, and necropsies on typhus patients reveal lesions of a diffuse leptomeningitis not unlike those of a rapidly coursing case of general paralysis.

The constant presence of a lymphocytosis is proof of the meningitic involvement.

III. Postexanthematous Neuritides and Polyneuritides.

Motor, trophic and sensory disturbances of the peripheral nerves may occur during, or after, the course of typhus. The writer has noticed such disturbances in fifty-five cases. The position in bed, trauma sustained during excitement of the delirium, and similar factors may determine the site and nature of the neuritis.

Subjective as well as objective symptoms may be prominent. Pains in the legs may produce functional disturbances in gait. The circumflex nerve is frequently involved, often owing to pressure of the shoulder on the bed. Mechanical restraint during delirium frequently results in an ulnar neuritis. Various inclemencies, including cold, heat and humidity, may be regarded as adjuvant causes.

Spasticity may result from pain and consequent disuse. The writer has also observed paresthesias, objective cutaneous disturbances of sensibility and modifications of electric reactions.

Bacaloglu has described a case of paralysis of the soft palate identical with diphtheritic paralysis.

The writer enumerates a variety of cases which have presented themselves at his clinic; one showed an atrophy of the infrascapular muscles; another a bilateral facial neuritis (which he calls diplegie faciale nevritique). He has observed three such cases, one not showing itself until nine months after the exanthem.

The etiologic factor is evidently toxic. But the toxins elaborated, though not yet known, are evidently carried by the vessels, and thus arise the vascular lesions such as phlebitides, and arteritides, which easily explain the myalgias and subsequent gangrenes. From the vessels the lesions extend to the peripheral nervous tissues, and we thus have the various neuritides and polyneuritides already described.

JONES, Detroit.

ON THE GENESIS AND PHYSIOLOGICAL SIGNIFICANCE OF SPASTICITY AND OTHER DISORDERS OF MOTOR INNERVATION: WITH A CONSIDERATION OF THE FUNCTIONAL RELATIONSHIPS OF THE PYRAMIDAL SYSTEM. F. M. R. WALSHE, M.D., TEMPORARY MAJOR, R.A.M.C., Brain **42**:1 (April) 1919.

In a previous paper, Walshe concluded from a study of certain of the reflex phenomena seen in the lower limbs in spastic paralysis, that "analysis of the phenomena of spastic paralysis of the lower limbs in man demon-

strates the close analogy existing between the physiological principles of motor innervation in animals and in man."

The author resumes the subject and discusses spasticity and decerebrate rigidity, Förster's theory of the origin of spasticity, the proprioceptive system of the cerebellum, associated movements in hemiplegia, three components in motor innervation, tremor-rigidity syndrome of paralysis agitans, other organic cerebral lesions, and cortical lesions and spasticity.

In the author's previous paper evidence was advanced that in spastic paralysis of the lower limbs there were two clinical types, the extended and flexed; and that in hemiplegia or in any spastic paralysis due to a lesion confined to the pyramidal system, the extended type was constant irrespective of the degree of pyramidal involvement. Walshe believes that both anatomic and physiologic considerations indicate that decerebrate rigidity is dependent on the removal of a crossed cerebral control.

As to the special position of the upper limbs in man, the view advanced in the present paper is summarized by the author as follows: "The spasticity of spastic paralysis as seen in hemiplegia and in paraplegia in extension is analogous with the decerebrate rigidity of Sherrington. It is, therefore, an expression of the activity of the proprioceptive system taken as a whole. This system induces a tonic reflex, which, unmasked in the decerebrate animal, has been shown to possess a significance of a postural reflex—reflex standing. In man also we see in hemiplegia a rigidity which has the same significance. However, in this instance, the upper limb shows typically an attitude of flexion. The hypertonus is resident in the limb flexors, not in the extensors as in the lower limbs. This may be traceable to the altered function the upper limb exercises in man, and to its complete lack of the locomotion function."

Walshe criticizes Förster's theory of the origin of spasticity and states that in each element Förster's hypothesis is incompatible with the facts.

Relative to the proprioceptive system and the cerebellum, he summarizes his views as follows: "Viewed in the light of Jackson's teaching and the physiological researches of Sherrington, there appear good grounds for regarding the cerebellum as an important link in the reflex arc subserving the rigidity of hemiplegia. We may, then, look for the 'complimentary inverse' of spastic paralysis of cortico-spinal origin in lesions involving the cerebellum. Both clinical and experimental observations demonstrate how completely these lesions may produce such a contrasted picture. In that the cerebello-rubrospinal path appears to be the efferent limb of the proprioceptive reflex arc, we may regard the motor disorders following its inactivity, which cerebellar disease entails, as an example of extrapyramidal disease."

Walshe ventures to suggest an analogy between the associated movements of hemiplegia and experimental red nucleus reactions. He says "if, as in hemiplegia, one corticospinal path is out of action, then control over the crossed postural reflex is in abeyance, and on voluntary movements of the intact path of the musculature, especially forceful movements, such as demand widespread synergic fixation, there occurs the usual bilateral postural adaptation through the action of the intact proprioceptive reflex system. Freed from cerebral control on the paralyzed path of the body, this is manifested as a crude, associated movement which thus consists in a transient modification of the spasticity."

The author criticizes Holmes' and Wilson's views of the cause of tremor and rigidity, characteristic of paralysis agitans and certain basal and midbrain lesions. He says that "we may conclude that the view attributing the

tremor and rigidity under discussion to uncontrolled cortical action is not established, and is contraindicated by such knowledge as we possess of the normal activity of the motor cortex."

Concerning the theory elaborated by Ramsay Hunt to account for the paralysis agitans syndrome, he states that the hypothesis is quite incompatible with what we now know of the physiology of such reflex acts as progression and muscle tone and, therefore, the theory of the functions of the corpus striatum, erected on the localization of these symptoms to a degeneration of this structure in a case of juvenile paralysis agitans, cannot be entertained.

Finally, he states that from clinical and experimental sources come several indications, as yet imperfectly understood, that a pure cortical lesion of the pyramidal system often does not produce spasticity as an accompaniment of the loss of power. They suggest the possibility of an interruption of another descending path than the corticospinal in the production of spasticity. Such a path need not necessarily be cerebrospinal.

POLLOCK, Chicago.

THE PATHOLOGICAL EXAMINATION OF FORTY INTRACRANIAL NEOPLASMS. J. GODWIN GREENFIELD, M.D., M.R.C.P., B.Sc., Brain **42**: 29 (April) 1919.

Greenfield reports the examination of a number of intracranial neoplasms from the pathologic laboratory of the National Hospital for the Paralyzed and Epileptic. The study is purely pathologic and no attempt has been made to link together the clinical symptoms and the pathologic findings. The chief object of the report is to make an attempt to clear up some of the anomalies of nomenclature and to discuss in relation to the findings in the cases examined, the nature of certain groups of tumors peculiar to the nervous system. The paper does not lend itself well to abstracting, and should be read in the original form.

Of interest is the analysis of the relative proportion, age incidence and rate of growth of the various forms of tumor encountered in this series. Tuberculomas total 5 per cent., and sarcomas 5 per cent., while granulomas account for only 2.5 per cent. The author states that this percentage obviously has no relation to percentage of incidence, but is due to the low rate of mortality in the granulomas and tuberculomas, and to the fact that sarcomas are more usually encountered in general than in neurologic hospitals. The group of endotheliomas includes 25 per cent., and perivascular sarcomas 7.5 per cent. In all, mesoblastic, intracranial tumors form 40 per cent. of the series. Gliomas total eleven, of which three are multiple. There were three neuroblastomas and one ganglioneuroma. Thus, glia and nerve cell tumors form 37.5 per cent. Lastly, there were six cases of acoustic nerve tumors, comprising 15 per cent. of the series.

While generalizations from such a limited number of cases are unjustifiable, the author deems it worth while to note the age incidence and duration of symptoms of the various forms of tumor. Both cases of tuberculoma were in young subjects, and gave a fairly short history. The sarcomas occurred one at the age of 11, and one at the comparatively advanced age of 54. In these cases the history was a matter of a few months. All the cases of endothelioma occurred after the third decade, with the exception of the two cases of myxo-endothelioma which occurred in children of 11 and 13, respectively. In these patients the symptoms appeared three and four years before admission to the hospital. The other endotheliomas gave histories dating back from

six months to six years. In the perivascular sarcomas, the author concludes that we are dealing with more malignant tumors but in one case the symptoms appeared probably five and certainly three years before admission. The gliomas of the series all gave a very short history, amounting in only one case to two years before admission and in the others to six months at most. The tumors of the acoustic nerve had histories of moderate length—the shortest, six months, was in a case of multiple tumors; the longest was two years.

No attempt will be made to summarize the views of the author on the nomenclature and classification of intracranial tumors because such a summary cannot be made and do justice to the author's opinions.

POLLOCK, Chicago.

THE PSYCHONEUROSES AND INTERNAL SECRETIONS. I. S. WECHSLER, Neurol. Bull. **2**:199 (May) 1919.

The author expatiates on the as yet unsolved problem of the treatment of the psychoneuroses and points out how little we really know about it, despite the fact that various attempts have been made to cope with the subject. He finds that most efforts are directed at the problem after it has become one, and he points to the internal secretory disorders as a possible method of attack before its incipiency. In the attempt to give a possible physical basis to the psychoneuroses and looking for a "little more solid ground to stand on" he turns to the internal secretions for an explanation of the symptoms in some cases. He does not lay claim to any originality but merely tries to correlate a few well-known facts with other facts equally well known. He calls attention to the danger of overestimating the importance of the "facts" of internal secretions, since we as yet know so little about them. Nevertheless, he thinks that we have before us a fruitful field for investigation.

After reviewing briefly the knowledge that has thus far been accumulated, he tries to correlate in a general way the symptoms of early disturbance of the glands of internal secretions and the common symptoms of some of the psychoneuroses which bear very close resemblance. Then he quotes two typical cases of psychoneurosis, another of anxiety neurosis, and one of hysteria, in all of which he found marked signs of disturbances of the pituitary and in which treatment along that line gave favorable results. One obsessional case and two of marked anxiety he quotes as having shown distinct thyroid symptoms, treatment for which cleared up the neuroses. One case is correlated with the suprarenals because the history of the neurosis bears close resemblance to that of an "early Addison," another one is linked with the sex glands and, finally, one case is put into the class of general vegetative nervous system disturbance—the vagotonia of Eppinger and Hess.

Although the author is aware of "the great importance of environmental influences of social maladjustments, of psychic and emotional conflicts," he seeks in "disturbance of the glands of internal secretions a source of rational therapy" for some of the cases of psychoneurosis and tries to put them definitely in a group which might well be amenable to treatment. One cannot, of course, quarrel with the author for his attempt, and he is careful to point out that success in treatment cannot be cited as proof of cure, inasmuch as it is difficult to dissociate the influence of the personality from the "effect" of a drug, but it is well to emphasize the meagerness of the facts at our command and the great danger of speculation which the fascinating study of the internal

secretions invites. We must wait for more accurate knowledge of the physiology and pharmacology of the glands and for more certain means of estimating disturbances before we shall be able to say definitely that a given psychoneurosis is the result of this or that glandular syndrome.

WECHSLER, New York.

TETANY IN THE EUNUCHOID: REPORT OF A CASE. H. W. WOLTMAN, J. Nerv. & Ment. Dis. **50**:433 (Nov.) 1919.

Woltman reviews briefly the literature on tetany, and reports a case because it has certain unusual features and because it is related to endocrine disorders of another source. The family history revealed convulsions and laryngospasm in various members of the family during childhood. A history of migraine was obtained and one brother developed a condition which strongly suggested an angioneurotic edema. The patient, aged 36, a barber, had had general convulsions beginning at the age of 1 and disappearing spontaneously at the age of 2. From the fourteenth to the sixteenth year he had had inflammation of the bowels associated with vomiting. At 21 a herniotomy and appendectomy were performed; after this he was perfectly well for a period of eight years. He then married. His wife has never been pregnant. Seven years prior to his application to Dr. Woltman for examination he noticed that his legs became easily tired; this condition persisted until one day his legs jerked up like a horse with "spring-halt." In the fall of 1915 the spasms appeared with increasing intensity, frequently five or six times a day. He had attacks which caused his mouth to remain wide open from thirty to forty minutes at a time; on other occasions trismus was present. The convulsions diminished in frequency, and in January, 1916, the patient was able to get about with the aid of crutches. His legs then began to swell and in August, 1916, they were two or three times their normal size. He also had edema of the abdominal wall and the back, and probably some ascites. In September, 1916, he suddenly cried out, lost consciousness and remained unconscious for three days. His legs became flexed at an angle of about 45 degrees and remained in this position. He lost weight very rapidly, so that one's fingers could be placed around his thigh; his face had a puffed-out appearance, and he was almost completely bald. The skin was dry and scaly "like a fish." The right testicle on examination was found to be flabby and small; the left was absent as the result of an orchidectomy which had been performed for tumor.

Woltman describes certain findings in his examination which he does not adequately explain on the basis of a tetany or other pluriglandular disturbance, and a report of postmortem is missing. Among these findings are a Babinski reflex, Chaddock's sign, a Rossolimo reflex and an ankle clonus on the right side, and a pallor of the disks suggesting a primary optic atrophy. He describes, in the course of the patient's illness, a number of attacks of tetany. Medication was of no avail, and the patient died March 3, 1917, greatly emaciated.

The author reviews some of the clinical features of tetany and points out the endeavor to identify more definitely the chemical nature of the substance at work, referring to Fuch's reference to ergotism, and to Eppinger's reference to histidin chlorhydrate. He calls attention to the work of MacCullum and

Voegtlin in relation to calcium deficiency. He quotes the views of Wilson and others relative to the ammonia content of the blood, and reviews Koch's theory.

He interprets the abnormal dryness and scaling of the skin in his case as suggesting thyroidal features, the presence of pigmentation noted during one of the exacerbations as of possible suprarenal origin and, finally, the direct evidence of disturbance of the gonads in the development of a testicular tumor with the clinical picture designated variously as pseudohermaphroditism, eunuchoidism, etc.

POLLOCK, Chicago.

CONTRIBUTION A L'ETUDE DE LA PATHOGENIE DU TABES.

ODILON GALLOTTI and SERGIO AZEVEDO, Rev. neurol. **26**:489 (June) 1919.

The pathology of tabes is one of the most frequently discussed problems in neuropathology. Advances are being made, however, in spite of disagreement of opinion; Schaudinn having conceived the idea that sleeping sickness is caused by a form of spirochete, Spielmeyer goes a step farther, contending that since syphilis is a spirochetosis, there must be some close relationship between sleeping sickness and general paresis of the insane. This in turn has led him in his studies to the opinion that tabes due to the trypanosome (in animals) is identical with syphilitic tabes in man. He founds this opinion on his experiments with dogs into whose subarachnoid spaces he injected the trypanosomes. By the Marchi method, eighteen out of forty-two animals showed degeneration of the posterior spinal roots, of the optic nerve and of the sensory branch of the trigeminus. Clinically, however, the animals showed no symptoms other than diminished reflexes of the fore legs.

The degenerative processes seem to show a preference for the cervical segments of the cord. The lesions in the peripheral nerves, however, are not to be distinguished from those produced by lead, alcohol or pellagra.

The chief resemblance, pathologically, between trypanosomic tabes and that of man is that both choose the focus where the posterior root pierces the pia mater. Spielmeyer's experiments on dogs prove conclusively the correctness of Schaffner's earlier contention that the cord lesion is secondary to the radicular lesion.

Spielmeyer's results led the writers to experiment on three dogs. Not being able to obtain a special strain of *Trypanosoma brucei,* they had recourse to *Trypanosoma gambiense* and *T. cruzi* which seem to have a special affinity for the nervous system. The writers describe their technic of inoculation. Two of the three dogs died; twenty-five hours later they found the trypanosoma in the blood of the animal that survived the experiment. At the end of three months they killed this animal, removed the brain and cord, stained sections in Mueller's fluid and by the Marchi method; nothing startling being discovered, they tried the Weigert method and found advanced signs of nerve degeneration in the lumbar region. They found, particularly, fibers that had lost the myelin sheath. In the cervical region, they found occasional lesions in the anterolateral column and constant changes on either side of the posterior root zone. Sections of the dorsal cord showed small lesions.

They conclude with the statement that their work agrees in its results with that of Spielmeyer.

JONES, Detroit.

THE PATHOLOGICAL CHANGES IN THE CEREBELLUM IN ACUTE ANTERIOR POLIOMYELITIS. Hubert S. Howe, Neurol. Bull. **2**:261 (July) 1919.

From the review of the literature, Howe found that little attention has been paid to pathologic changes in the cerebellum. Clinically the diagnosis of cerebellar affection is usually diagnosed by the ataxia, but as others have pointed out and as Howe, himself, says, there are many other locations in the cord and brain which may account for the ataxia. The author cites a few case histories reported by other observers to show the clinical history of poliomyelitis with cerebellar changes, but one of the cases reminds one more definitely of a cerebellar apoplexy than of an encephalitic process.

Howe quotes many authors to show that pathologic changes in the cerebellum are absent in poliomyelitis, are usually not marked, and are often difficult to find when present. Some authors have described mild perivascular infiltration and changes below the granular and Purkinje cell layers; others described the lesions as thrombosis of the terminal arterioles; still others found pial lymphocytic infiltration and slight hemorrhages.

Howe's findings are: Macroscopically, the cerebellum appears edematous and congested. Microscopically, the cerebellar pia frequently shows diffuse inflammatory changes, more marked than in the cerebral pia and closely resembling that of the cord. Edema, hemorrhage, congested vessels and exudated cells separate its layers, but the picture differs from ordinary meningitis in that there is no fibrin and no exudate on the pial surface. The infiltrating cells are lymphocytes, few large mononuclears, polyblasts and polymorphonuclear leukocytes. The vessels are congested, but no thrombosis was ever encountered. The most striking change is cellular infiltration in the Virchow-Robin lymph spaces, which, however, is less common than in the cord and also differs from the latter in that the white matter presents the most frequent sites.

The ectodermic changes occur both in the white and gray matter of the cerebellum. Focal areas of cellular accumulation are seen, which are less intense than in the gray of the cord, and do not seem to bear any relations to blood vessels or nerve cells. The Purkinje cells in the infiltrated areas only show degenerative changes. As in the cord, the infiltrating cells are made up of lymphocytes, polyblasts, ameboid neurologic cells, plasma cells and a few polymorphonuclears. The Purkinje cells may show slight cloudy swelling but are usually normal. The changes in the white matter are more marked than in the gray, and affect mostly the deeper white substance of the peduncles and the central white.

The author concludes that, inasmuch as the poliomyelitis virus has little affinity for the cerebellar nerve elements and the changes are mostly confined to the white substance, "the clinical symptoms referable to the cerebellum are in reality due to interference with the function of its connecting pathways and not to the destruction of nerve elements."

Wechsler, New York.

A CONTRIBUTION TO THE STUDY OF THE PATHOLOGY OF HUMAN AND EXPERIMENTAL POLIOMYELITIS, BASED ON CASES OCCURRING DURING THE EPIDEMIC OF 1916 IN NEW YORK CITY. Hubert S. Howe, A.M., M.D., J. Nerv. & Ment. Dis. **50**:409 (November) 1919, continued from **48**:235.

Hubert S. Howe continues with the protocols of cases of experimental poliomyelitis, and discusses his findings which he summarizes as follows:

1. The tissue changes in acute anterior poliomyelitis are of three distinct types—those which predominantly affect the ectodermogenic elements, those which predominantly affect the mesodermogenic elements, and those which affect both these elements about equally.

2. The ectodermogenic type of tissue change in the central nervous system of man is polymorphous; that is to say, the cellular constituents derived from the ectoderm show no one form of degenerative process. The reaction in the ganglionic cells and neuroglia makes it possible to recognize no less than eight different forms in the degenerative process consequent to the poliomyelitis infection.

3. The degenerative changes in the ectodermogenic elements are so diverse as to make it impossible to conceive of them as phases in a single disintegrative process.

4. The mesodermogenic type of tissue reaction presents the more general reaction of the organism to the infection and manifests itself by uniform changes affecting not only the central nervous system, but also the lymphoid tissues throughout the body.

5. Changes in the elements derived from the ectoderm, neuroglia and ganglionic cells are, in the main, not secondary to or dependent on the changes in the mesodermogenic tissues, but represent a specificity on the part of the ectodermogenic elements in their reaction to the virus.

6. The susceptibility of the motor cells in general and the anterior horn cells, or their homologues in the brain stem, in particular, furnishes further evidence of the specificity of the poliomyelitis virus.

7. The possibility of such definite specificity in a given virus is demonstrated by the exclusively ectodermogenic reaction observed in the pathologic changes resulting from the feline epidemic which was coincident with the poliomyelitis epidemic of 1916.

8. While it is possible to conceive of certain conditions which might underlie a selective reaction of such a specificity, the determination of these conditions still remains to be disclosed by future work in this field.

9. In human poliomyelitis, the pathologic reaction to the virus is a mixed one, in which both mesodermogenic and ectodermogenic changes occur. It is undoubtedly true that the most severe impairment of function results from the changes in the elements of ectodermal origin, while it seems probable that the more transitory or even abortive disturbances of normal function are the result of changes almost, if not wholly, limited to mesodermogenic tissues.

POLLOCK, Chicago.

EARLY NEUROSYPHILIS ASYMPTOMATICA WITH REPORT OF OBSERVATIONS AND CASES. JOSEPH VICTOR KLAUDER, M.D., Am. J. Syphilis **3**:513 (Oct.) 1919.

The author expresses the belief that probably in all cases of primary and secondary syphilis the nervous system is invaded by the treponema. In support of this view he has collected from the literature and from his own observations clinical and laboratory evidence.

Spinal fluid studies on 214 cases of primary syphilis by various syphilologists are cited, only forty-seven of which showed an abnormal spinal fluid content. It is to be noted that the abnormality usually consisted of a slight increase in the cell content and that there was a wide difference of opinion as to what constituted a normal cell count. Only two cases showed a positive spinal fluid

Wassermann reaction. The percentage of neuraxial involvement found in secondary syphilis is given as from 10 to 90 per cent., the number of cases studied not being stated.

Treponema pallidum was demonstrated in the spinal fluid in fourteen cases of primary and secondary syphilis; the spinal fluid in these cases was otherwise frequently normal; the subjective and objective phenomena were frequently absent.

The clinical manifestations of early neurosyphilis are reviewed, the symptoms virtually always being referable to an involvement of the meninges. To the usual three clinical types of meningeal involvement he adds a fourth called the asymptomatic group, which as the name indicates, lacks subjective or objective symptoms; but it possesses conspicuous spinal fluid evidence.

Of the cranial nerves, the eighth is the most frequently involved. The others involved, in order of frequency, are second, seventh, third, fourth, fifth and sixth.

In a series of twenty-five cases the author made an initial examination of the spinal fluid in the untreated eruptive stage of syphilis and a second one after the administration of four intravenous injections of arsphenamin 0.5 gm. at from seven to ten day intervals, this being the only treatment given. In every case there was an accentuation of the eruption, and a Herxheimer reaction after the first treatment. In one case a negative spinal fluid became positive after treatment; in another, in addition to spinal fluid changes, clinical evidence of neurosyphilis in the form of a severe headache developed, but disappeared on further treatment. The latter phenomenon is ordinarily known as neurorecidive. The term neurorecidive and the Herxheimer reaction are defined, and a brief review of the literature on the subject is made.

The author cites his own observations in substantiation of the view expressed that neurorecidive is an expression of the Herxheimer reaction. The neurorecidive phenomenon is divided into two stages—the laboratory or asymptomatic, and the clinical or symptomatic. The former is styled "neurosyphilis provocativa asymptomatica"; the latter, "neurosyphilis provocativa symptomatica."

The Herxheimer reaction is regarded prognostically of unfavorable significance. Its excitation should therefore be avoided in the treatment of syphilis by preceding the arsphenamin therapy with a course of mercury because of its milder spirochetacidal properties.

Just criticism is made of the insufficient and exclusive use of arsphenamin in the early treatment of syphilis.

HAINES, Philadelphia.

IRREGULARITY IN A PSYCHOLOGICAL EXAMINATION AS A MEASURE OF MENTAL DETERIORATION. SIDNEY L. PRESSEY, PH.D., and LUELLA W. COLE, A.B., J. Abnorm. Psychol. **13**:285 (Dec.) 1918.

Pressey and Cole present a comparative study of the amount of "irregularity" or so-called "scatter" between a group of feebleminded and psychotic subjects all showing mental ages from 8 to 12 years. "Irregularity" is used as a term to denote the sum total of the variations from the original point scale for norms. For instance, the feebleminded score is characterized, not only by its low level, but also by its regularity and the absence of such discrepancies as are brought out in psychotic persons; namely, the inability to pass certain tests, together with the ability of passing others which are respectively far below and above the mental age as determined by the entire scale. The group tested comprised 158 defectives, sixty-seven schizophrenics

and fifty-five chronic alcoholics. The average "irregularity" of the defectives totaled 17.5; the schizophrenics 20.1 and the alcoholics 22.3. That there was considerable overlapping is evidenced by the fact that 25 per cent. of the feebleminded revealed as much "scatter" as did the average praecox patient. From an analyses of the results the authors then selected five differential tests. These not only emphasized the preponderance of "irregularity" in the psychotics (6.9 for the schizophrenics and 8.2 for the alcoholics as against 4.7 for the defectives), but also markedly decreased the overlapping. In only 10 per cent. of the members of the feebleminded group was the "scatter" as great as in the praecox subjects, and in only 2 per cent. as compared to the inebriates. "Irregularity" in mental diseases may be determined by a variety of factors, such as temporary negativism, retardation or delusional trends. It therefore measures more than merely the amount of mental deterioration. A point of some practical importance lies in the suggestion that pre-existing and basic intelligence deficiency may be suspected in those psychotics who test low but evenly, i. e., without "irregularity."

The authors realize that it would not be justifiable to draw far-reaching conclusions from the limited number of subjects tested. Furthermore, there are tremendous obstacles that stand in the way of applying a pure intelligence scale to abnormal mental states. It is somewhat doubtful if the various factors in the psychoses which might give rise to "irregularity" can ever be standardized. Even in the so-called deteriorating psychoses, such factors must vary greatly from time to time and in the same patient. For instance, in a group of praecox patients one may expect much individual variation in the affective sphere, in the domain of the will and in the ideational realm within comparatively short periods of time so that the index of "irregularity" will be found to be far from constant, even in the same subject.

STRECKER, Philadelphia.

A CASE OF TUMOR OF THE CORPUS CALLOSUM AND FRONTAL LOBES. C. C. BELING, M.D., and H. S. MARTLAND, M.D., J. Nerv. & Ment. Dis. **50**:425 (November) 1919.

Beling and Martland report a case of a male, white, 54 years of age who, at 30 years of age, suffered for a few days from vertigo. Jan. 23, 1914, some hypersensitiveness to light and diminished vision were noted. In April, 1916, considerable loss of memory was revealed, and May 19, 1916, he had a sudden convulsive attack which began with twitching of the right arm, hand and leg. May 24, 1916, he had recovered sufficiently to resume his work. A week or two later he was talking and acting in a silly manner. Examination revealed a bilateral optic neuritis, and the gait was unsteady and somewhat ataxic; "clonic perseveration" (Liepmann) showed markedly in his handwriting. Involuntary urination and defecation were present. Because of the disturbances of association resulting in apraxia, chiefly of an agnostic, ideomotor, aphasic and agraphic character, a clinical diagnosis of tumor of the corpus callosum and frontal lobes was made. Automatism and defects of volition (lack of initiation of movement ideas) were explained by an involvement of the left prefrontal lobe. A postmortem examination made eight hours after death, on July 3, 1916, revealed a tumor occupying the inner halves of both frontal lobes, the genu and the anterior one-third of the body of the corpus callosum. The tumor was infiltrated in character, fairly well circumscribed but not encapsulated. It presented the gross characteristic of a

telangiectatic, infiltrative, noncircumscribed glioblastoma. It was composed of proliferating neuroglia cells presenting a whirl-like, radiating and wavy arrangement. The greater part of the tumor presented extensive retrograde changes, especially necrosis and hemorrhage.

POLLOCK, Chicago.

SCIATICA WITH MUSCLE HYPERTROPHY. CAPT. C. PASTINE (Chief Neurological Service, Field Hospital 227), Riv. di patol. nerv. **24**:46, 1919.

The author says that Roccavella added eight new signs of sciatic neuritis to the twenty-five already known as a result of observation of many cases during the war. The author adds still another, namely, hypertrophy of muscles instead of the usual atrophy seen in sciatica. Careful reading of the ten case histories presented shows that all of them are open to serious criticisms as to diagnosis. No sensory examinations are detailed; the neurologic signs are scant. Achilles' reflex is present and the patellars are active in several and clonus in one. The author speaks of tenderness and pain in the gluteal, and thigh and calf hypertrophies. Many of the ten patients had abnormal attitudes caused by spinal disability or deformity or compensatory scoliosis from hip and knee joint involvement. The author's finding of hypertrophy of muscles in sciatic neuritis is seriously questioned. During my service as neuropsychiatrist to the American forces in Italy during the war, I was ordered on detached service to the author's military hospital and directed the neurologic service during the author's illness. I saw many of the cases of "sciatica" so-called. No sensory examination worthy of the name was recorded. The hospital did not boast of a roentgen-ray apparatus. Many of the cases were gluteal or other types of myositis; many were surely more or less obscure joint and spine conditions. A few that I saw were true sciatic neuritis. None of these had hypertrophy of muscle. One case of "sciatica" was a tabetic. Of the ninety-eight cases of "sciatic neuritis" seen by the author, ten presented muscle hypertrophy. All of these conditions came on after prolonged exposure to rain and snow and cold in the mountain trenches which this hospital drained. The author admits that many of the cases showed cyanoses, thermic and vasomotor symptoms in the extremities, accompanying so-called "trenchfoot" and not sciatic neuritis. The author's statement that true sciatica is accompanied in many cases by muscular hypertrophy directly dependent on the neuritis is open to grave doubt.

OSNATO, New York.

AN ACUTE PRISON NEUROSIS OF THE ANXIETY TYPE. N. S. YAWGER, M.D., J. Nerv. & Ment. Dis. **50**:319 (October) 1919.

N. S. Yawger analyzes this type of neurosis in a brief and comprehensive manner. He states that it is more common in penitentiaries to which, generally speaking, felons are sentenced, than in the smaller jails where those only guilty of misdemeanors are interned. About one third of the convicts coming up for pardon show nervousness to a greater or less extent and in one tenth of these offenders the subjective symptoms are so suggestive as to be almost diagnostic. Although hoping for pardon is productive of a more intense neurosis than is the approach of the expiration of a minimum sentence, on obtaining freedom the pardoned person recovers more promptly. The subjective evidence of the disorder appears in the form of restlessness, decreased power of mental concentration, irritability, hypochondriac and introspective manifestations, dreams of a disturbing nature, often broken by dis-

tressing insomnia, dyspepsia, genito-urinary manifestations, cardiac irregularity or respiratory disturbances; usually, there is an appreciable loss in weight and in smokers, an increased use of tobacco.

In the consideration of the nature of this disorder the author believes that undoubtedly the reactions of this anxious period are the result of important factors aside from the sexual sphere. Such factors include the reactions to a situation wherein the prisoner knows that detainers have been lodged against him and that he will be homeless, and the certain knowledge that he will be an unwelcome member of society.

POLLOCK, Chicago.

CONCERNING HAMLET AND ORESTES. JOHN T. MACCURDY, M.D., J. Abnorm. Psychol. **13**:250 (Dec.) 1918.

Must "autistic thinking" follow certain channels that are common to all individuals, no matter how far separated in time, place, or civilization?

Hamlet and Orestes figure in stories derived from sagas of the early Northmen and pre-Greeks. And yet there are striking likenesses in the plots of their stories and in the character and "madness" of the heroes. The type of insanity is given as dementia praecox because the following symptoms appeared: seclusiveness, hallucinosis, ideas of reference, scattered speech, inappropriate affect, filthiness, and "queer behavior with sudden, unexplainable assaultiveness." With these symptoms are linked ideas of incest and death. The author's conclusion is that the inspired dramatist in dealing with a hero of incestuous tendencies, is forced by his very genius to paint about his hero the clinical picture of dementia praecox, for in all men there is a "basic, unconscious connection between ideas of a given type" and this disease.

It is too much to ask that the big idea which underlies this article and makes it stimulating shall carry the assumption that there is no dementia praecox without an Oedipus complex or to depend on a definite diagnosis of the disease of a mythical hero who is Shakespeare's Hamlet mixed with many Norse pre-Hamlets.

BOND, Philadelphia.

A NEW CONCEPTION OF EPILEPSY. P. HARTENBERG, Presse méd. **27**: 664 (Nov. 8) 1919.

The author offers a new conception of epilepsy. He gives as the cause of the disease *inhibition* of the centers of the cortex rather than stimulation. As proof he cites the frequency of brief amnesia and vertigo; in other words, the petit mal compared to grand mal. These are cessations of functions rather than increase of them. He goes further—he says that grand mal is only the petit mal plus convulsions plus coma. His formula is: amnesia = unconsciousness; vertigo = unconsciousness plus falling; grand mal attack = unconsciousness, plus falling, plus convulsions, plus coma. He asks whether convulsions may not also be the result and product of inhibition, and cites the numerous comas which are accompanied by convulsions. In short, inhibitions of the higher cortical centers may set free muscular discharges. Only two conditions are necessary, and these are both present in epilepsy, namely, that the inhibition be brusque and that it be profound. The analogy of the convulsive movements to those produced by a lesion of the corticothalamic tracts (forced laughing and crying) stimulates Hartenberg to believe that the convulsions of epilepsy are due to absence of cortical control of the lower centers.

By experimenting with the effect of strychnin, Dr. Hartenberg found that it notably diminished the symptoms rather than increased them, which lends plausibility to his theory.

In short, epilepsy is not a convulsion, but a state of unconsciousness. It is due to inhibition of the activities of the upper cortical centers rather than to irritation of the motor centers.

This article is interesting and well worth a careful perusal.

KRAUS, New York.

THE INSIGNIFICANCE OF LOCAL DISTURBANCES IN THE PRODUCTION OF CONSTITUTIONAL NERVOUS SYMPTOMS. CHARLES MCKENDREE, Neurol. Bull. **2**:253 (July) 1919.

The author bases his conclusions on his observation of some 3,500 neurologic cases, the vast majority of which were so-called functional cases. Bearing in mind the great emphasis that is laid on focal infections, reflex irritation, visceral disturbances, etc., which have been so frequently incriminated as the causes of obscure psychic complaints, he tried to study a number of cases. Wherever there appeared the slightest indications, he referred the patients to the dentist, oculist, nose and throat specialist or gynecologist, etc. Frequently each specialist found something which he was sure was the cause of the headache, the pain or indigestion. The patients underwent palliative, corrective or radically operative treatment, but despite that their chief symptoms remained. He quotes five cases to illustrate the futility of treatment in patients who showed no general physical or neurologic signs, but who were supposed to suffer from some obscure "cause."

Without trying to minimize the importance of special causes in the production of general symptoms, McKendree revolts against the faddist tendency in medicine to run away with oneself as focal infectionists, for instance, do when they try to explain every vague symptom, or any condition from bunion to brain tumor, by an apical abscess or ungeometrical tonsillar crypt. He agrees that there are cases, here and there, which benefit from special attention, but those cases show definite symptoms which can be well correlated with the underlying causes. The vast majority of constitutional nervous symptoms, however, have no such etiology, and the few who do show improvement with special attention would show it just as well on general hygienic or other treatment. Besides, most of the patients do not respond to such treatment, and what is more important, they become confirmed neurotics because of the radical attempts at cure. The author, therefore, pleads for a little more common sense in the evaluation of symptoms and the bridling of the radical tendency to seize on insignificant and unsubstantiated conditions as the root of all constitutional nervous disorders.

WECHSLER, New York.

THE CEREBROSPINAL FLUID IN HERPES ZOSTER, AND THE RELATION OF HERPES ZOSTER TO SYPHILIS. W. H. BROWN, M.D., CAPTAIN, R.A.M.C., and DR. B. DUJARDIN, Brain **42**:86 (April) 1919.

The authors report the cerebrospinal findings in twenty-five cases occurring in Captain Brown's experience, and sixteen cases observed by Dr. Dujardin. The cytologic estimations showed striking, inconstant and puzzling results. In the majority of cases, twenty-eight, there was some degree of lymphocytosis, but this varied enormously from normal to an intense lymphocytosis (470 per

cubic millimeter). In ten cases the counts registered over 50 cells per cubic millimeter, in ten, the counts registered within normal limits. In the majority of cases there was a slight increase of albumin. In only one was there a clear, decided ring obtained by the Nonne-Apelt method of testing for globulin.

Relative to Dr. Dujardin's statistics, the striking feature is the frequent incidence of herpes zoster among syphilitic subjects. Of 1,200 cases of syphilis in one stage or another, nine developed herpes zoster. Among 13,000 other patients only ten cases of herpes zoster was seen. The authors state that it would appear that a predisposition to herpes zoster existed in syphilitic subjects. They also point out the fact that in their cases herpes zoster in syphilitic subjects had a predilection for the lumbar and sacral ganglions. Conversely, in a case of herpes zoster the possibility of a prior syphilitic infection must be considered, more especially if the herpes involves the lower spinal ganglions.

POLLOCK, Chicago.

Society Transactions

THE AMERICAN NEUROLOGICAL ASSOCIATION

Forty-Fifth Annual Meeting, held at Atlantic City, N. J., June 16-18, 1919

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9. NERVOUS AND MENTAL DISEASE IN UNITED STATES TROOPS. PEARCE BAILEY, M.D., New York.

Statistical tables showing incidence as regards age, locality, social condition, nationality, length of service, methods of detection, etc.

DISCUSSION

DR. JOSEPH COLLINS, New York: I should like to speak of two points that were brought out in the paper by Dr. Bailey. The first is: There are in the United States a great number of potential nervous and mental diseases among the youngest and most active persons physically of the community. They are not recognized by the medical profession, which includes the neurologist. Therefore there must be something radically wrong with the relationship of the medical profession to the community. The second point is: There are in the community a large number of young men capable of earning their livelihood who yet must be classified as high grade imbeciles. So far I have not learned much from the war concerning the pathogenesis of nervous disease, but I have learned something concerning my relationship to the community and have experienced a decided quickening of my communal conscience. We have a larger work cut out for us than the diagnosis, interpretation and attempt at treatment of well developed disease, and that work lies in the field of cooperative medical pedagogy and preventive medicine. The medical profession of this country was unprepared to make the so-called neuropsychiatric examinations when war was declared, unprepared without intensive training to take care of neuropsychiatric cases when they were detected and when they resulted from war and the incidences of war. It is now less prepared than many nations to enter into the work of reconstruction from the neuropsychiatric point of view. The cause of those defections may be summarized in one sentence. The neurologist has never been properly taught in this country and the internist has devoted his energy to scorning the neurologist and treating with contumely the things he must now recognize. It is our duty as neurologists and psychiatrists to lead medical thought in this direction. We must convince the profession and the community that it is the man as well as the disease that must be treated; that the emotions which are an integral part of a man are bound up as closely through the endocrine system through the somatic nerves with disease as man is bound up in original sin. We must embrace our opportunity now to do missionary work, not among the uneducated but among the leaders of medical thought and endeavor in this country: first, by showing that the nervous system and its disorders is not a sterile field of medical research and labor; secondly, by making men capable of teaching neurology and psychiatry; thirdly, by not permitting a student who has not acquired the principles of neurology and psychiatry to enter the portals

of medicine, and fourthly, by recognizing that it is more important that a favorable medical atmosphere be developed than that hospitals for nervous diseases be built. It is our duty and our opportunity to develop that atmosphere.

10. SOME PSYCHIATRIC LESSONS OF THE WAR. Dr. Thomas W. Salmon, New York.
No discussion.

11. HYSTERIA IN THE LIGHT OF THE EXPERIENCE OF WAR. Lieut.-Col. Arthur F. Hurst, M.D., R. A. M. C., London, England.

This article appeared in the November issue of the Archives of Neurology and Psychiatry, page 562.

DISCUSSION

Dr. Morton Prince, Boston: I would like to say a few words in connection with Colonel Hurst's paper, and to pay a tribute to his great skill in curing his patients. But when he comes to the pathology that he has adopted from Babinski's teachings I am compelled to take issue with him. If it is said that hysteria is due to a psychic cause, we can all agree, for that is a well accepted fact, but when he says that hysteria is solely the result of suggestion, the conception in my view, is entirely inadequate and is based on a consideration of not only a limited number of types of hysteria but of a limited number of its manifestations and without regard to the fundamental underlying pathologic state. As the "accidents" or manifestations of hysteria Babinski recognizes only those phenomena which he avers are capable of being reproduced experimentally by suggestion and correspondingly can be made to disappear by suggestion. The essential symptoms that he recognizes as hysterical are: convulsive attacks, paralyses, contractures, tremors, choreic movements, troubles of phonation, respiration, sensibility and sensorial troubles. He defines hysteria in the following way: "Hysteria is a *pathological state* manifested through troubles which it is possible to reproduce by suggestion in certain subjects with a perfect exactness and which are susceptible of disappearing under the influence of persuasion (contra suggestion) alone."

Strange to say, however, after the formulation of this definition, we hear nothing more of the pathologic state—only an elaborate and clever exposition of its manifestations. This failure to keep in mind, on the part of Babinski, the conception of the pathologic state is the weak point in the edifice which he builds up. Dwelling only on symptoms, he fails to grasp the essential problem of hysteria, losing sight of the pathologic state which should be the goal or solution sought. He confines himself to certain physiologic phenomena and loses sight of the fact that many of the so-called troubles which are the manifestations of the "hysterical state" are pure mental stigmas. These are not included in his first group of pithiatic symptoms, i. e., they are not recognized as hysterical, though they can be induced in favorable subjects by suggestion. Necessarily, therefore, he fails to recognize that many of the cases which he makes use of to support his thesis were already in the "hysterical state" and manifested classic manifestations of hysteria. The cases that had been exposed to emotional trauma, he supposes, were free from hysteria until they were later the victims of suggestion. The consequence of this lack of vision is that when he comes to his data, to the cases which he cites to show that hysteria is not induced by emotion but only by suggestion, he naively describes cases in which every clinician ought to recognize that the patient

had already developed hysteria and had already exhibited classic hysterical symptoms of a mental sort induced by emotional trauma or mental stress and strain, before suggestion, even in his own opinion, had had effect.

The corner stone of Babinski's thesis, then, is that the hysterical accidents (pithiatism), very limited according to his conception as we have seen, are always the result of suggestion—autosuggestion or heterosuggestion.

I have no cause to quarrel with Babinski for insisting that hysterical phenomena can be suggested. Every neurologist has seen often enough examples of this in his practice, and, indeed, is aware that he himself has suggested such phenomena, intentionally or unintentionally. Nor can any one doubt that a large proportion of hysterical stigmas, particularly those following traumatisms, have been so suggested, generally unconsciously, by the examining physician. But that all have been so suggested, or what proportion have been so suggested, is another question. Given a certain abnormal state of mind, symptoms can be manufactured almost ad libitum. In such a state the symptoms can be educated, just as by reeducation they can be made to disappear.

But what is the underlying pathologic state that permits such suggestibility, and which Babinski passes over? And is this "state" induced by suggestion alone? This is the real problem. Every one recognizes at the present time that the "pathological state" of hysteria is an emotional dissociation of one or more of the normal integrated psychoneurologic systems manifesting itself by loss of certain mental and neurologic functions, on the one hand, and overactivity of certain other functioning systems, on the other. If you do not like the psychologic term "dissociation," you may use the physiologic term disintegration, or inhibition or repression. We are not now concerned with the mechanisms by which these phenomena are brought about. The question, then, becomes not simply whether the manifestations are due to suggestion and to suggestion alone, but whether the dissociation or disintegration is induced by suggestion and suggestion alone. Babinski devotes much effort to showing that emotion, contrary to the classical view, does not and cannot induce hysteria. He even goes so far as to maintain that "when the human soul is shaken by sincere, profound emotion, there is no longer a place for hysteria." If emotion, then, can be eliminated, no other agent, he assumes, is left but suggestion. In support of this thesis he contends that hysterical phenomena never appear at the moment of or immediately after the emotional shock, when the emotion is at is height, but that always "between the emotional shock and the presence of hysterical ("pithiatic") accidents there is an intervening phase, sometimes quite long, which Charcot called the "phase of meditation," during which autosuggestion or heterosuggestion have the opportunity to intervene and induce the accidents. As evidence he cites the observations of numerous writers who had the opportunity in this war to observe so-called "shell shock" at the front and behind the lines. Babinski seems at first to be supported by these observations, for it appears that those symptoms of hysteria which Babinski elects to call alone pithiatic or hysterical—that is the paralyses, anesthesias, etc.—rarely developed at the time of the emotional shock, but appeared after an interval, when the soldier had reached some place behind the lines.

A study of the reports, however, shows that the cases, apparently without exception, exhibited at the emotional period immediately following the shock, hysterical mental symptoms of a very marked character. Among the symptoms commonly described are: amnesias, hallucinations, deliria, inability to respond to questions, even when attempts were made to force a response, appar-

ent incapacity to perform a voluntary act, states of hebetude, stupor, confusional states, states allied to fugues, mental dulness, irrational states, tachycardia, tachypnea and tremor.

What are these, it may be asked, with the exception of the three last (which are only the physiologic manifestations of emotion), but states of dissociation with automatisms or unregulated and uncontrolled functioning of disintegrated psychologic systems? They are, from the modern point of view, typical and pure symptoms of the hysterical pathologic state. Amnesia, for instance, as already pointed out, is well recognized as a manifestation of dissociation. In these very cases, then, cited by Babinski in support of his thesis, we have evidence of hysterical manifestations induced by something else than suggestion, and we must remember that this cause is conditioned, at least, by emotion. It may also be pointed out in passing that these mental symptoms, following the discharge, may be brought within Babinski's own definition of hysteria in that they are capable of being produced experimentally by suggestion.

That certain symptoms like paralysis, anesthesia, dumbness, deafness, etc., do not appear until a later period may be a fact, as a matter of observation, but the real question is *why* do these particular symptoms appear later, while other and mental symptoms appear at the height of the emotional discharge? It is a question of the *why*. The fact that seems to have been brought out by observation during this war is that the hysterical state, manifesting itself by mental dissociation, can be induced immediately under mental stress and strain at the moment of the emotional discharge, while certain other symptoms, in the great majority of cases, develop only after a period of incubation.

Babinski seems to assume that because this latter class of symptoms develops only after a period of incubation they must be due to suggestion, particularly to heterosuggestion, although autosuggestion may be the genetic factor. To this assumption exception may be taken. That many hysterical stigmas—particularly the paralyses anesthesias, crises, deafness, numbness, etc.—have their genesis in psychologic factors, there can be no doubt, and I think that since 1885 there never has been any doubt. That we have all known for a long time. But this is far from saying that they are due to suggestion, whether autosuggestion or heterosuggestion. The genesis and the mechanism are far too complex for such a simple explanation. They may be called "defense reactions," if you like, according to one theory; but the psychologic mechanism of a defense reaction is complicated. This may be due in individual cases to mental conflicts, to subconscious mechanisms of different kinds, etc.

Babinski's fundamental error is in not recognizing that the hysterical state is one of functional dissociation and that any psychologic factor capable of producing such a dissociation, whether it be emotion or a conflict, is capable of producing hysteria. The mechanism by which individual symptoms is produced is another problem. It may be suggestion, as we all know, or it may be a very complicated mechanism which still requires solution.

Dr. Foster Kennedy, New York: Hysteria has been described as a dissociation of the personality, and Colonel Hurst has portrayed that dissociation. But he must have appreciated that such dissociated personalities were unstable, and I should like to have him say a few words about the disposal of such soldiers after cure—whether an attempt was made to educate each man to be a more stable personality, or whether disposal was made of him so that he was put in an environment where easy adaptation was made pos-

sible for him. Without giving this information, Colonel Hurst is apt to give too easy a solution of the problem. I am in entire agreement with him regarding the functional, emotional and "suggested" character of the so-called main gêlé described by Babinski as being produced by reflex neural physiologic block consequent on trauma. I came to the conclusion that probably every wound of the upper extremity was associated with a temporary defensive immobilization; the man feared to move his injured arm, and if he were asked if he could do so he would invariably reply in the negative. If the medical officer took such a statement at its face value, the seeds of hysteria were immediately sown. The improper suggestions which colored these pictures and produced the paralysis were almost always the result of incomplete medical knowledge on the part of the medical officer.

DR. D. J. MCCARTHY, Philadelphia: I might say, from the standpoint of the estimation of war risk, that this paper is of great value, and I agree with Colonel Hurst that the vast majority of the cases which he describes are purely hysterical. There is nothing new in any of the cases shown, and those of us with war experience during the last four years, could easily duplicate this group.

I am not willing to agree with the statement that this group of cases entirely eliminates the Babinski reflex group. In 1915, I saw some cases that appeared at first glance to be functional, but which proved, on further study, to have an added element not altogether organic—functional in a different sense from the purely hysterical. The reflexes were sometimes active, sometimes lost. The atrophy was sometimes of rapid development and associated with edema. This was particularly true of injuries near the shoulder joint and of the shoulder girdle. It was more common in pulverization of the humerus. This led me to examine a series of cases at the American Ambulance at Neuilly in 1915, immediately after they had received extensive injuries to the humerus, shoulder joint, etc. I found that there was sufficient evidence to indicate a reaction of the peripheral neuron, and its spinal segment, as shown in motor, trophic and reflex manifestations, to lead me to put the other cases into a separate group to which I gave the name of "metameric functional states." Even after recovery these patients present a condition of reflex disturbance, usually in the nature of increased reflex activity with easy fatigability, more marked in the affected side but often bilateral in distribution. Since 1915 I have seen these cases both in army and civil practice. To my mind they present a clear cut and distinct group, somewhat different from the Babinski group, but more limited and better defined. They can be explained by the reaction at a distance of the spinal segments of the cervical cord directly concerned with the sensory supply to the muscle groups, having their origin and insertion on the bone extensively injured.

There is another phase of the subject of great interest to me—the relation of war risk insurance to nerve injuries. In a majority of the peripheral nerve cases studied, a series of symptoms was added to the organic group, to which I gave the name "functional overflow." These hysterical or functional symptoms varied in extent, and could easily be differentiated from the organic symptoms. I may say that this functional overflow was seen not only in the nerve cases, but was as evident and as equally frequent, in the heart cases, gas cases and other visceral diseases. The internist, with his lack of interest in nervous diagnosis, usually overlooked this functional overflow to the detriment of the patient and of the army. One of the most important functions assigned to the staff in the institute at Vichy was to strip the incoming patient

of functional or overflow symptoms, before these symptoms became fixed, and to chart the residual organic symptoms for the benefit of the surgeons in the United States, whose duty it might be to operate, or to discharge the patient to civil life, with a pension based on percentage disability. Some highly efficient person saw fit to strip every field card of these valuable records at the port of embarkation for some reason perceptible only to an army type of mind.

Dr. Hugh T. Patrick, Chicago: I want to embrace the psychologic moment to say a word about Colonel Hurst's paper. His presentation was to me an illimitable joy because it was a demonstration of neurologic renaissance in England. At the opening of the war the English were less prepared than any other nation to understand, and consequently to treat, the war neuroses. From Colonel Hurst's presentation and from the reports emanating from his hospital, it is obvious that he has undertaken and is succeeding in a popular propaganda for education of the English medical public in the matter of the psychoneuroses, especially hysteria. That this is, or was, greatly needed is borne out by his categorical statement that nearly all of the orthopedic cases seen by a neurologist proved to be hysterias and that nearly all the military rheumatic cases at Bath were neurologically shown to be the same disorder.

But it is to be added that all of the matter so graphically presented by Colonel Hurst has already been embalmed in medical literature—most of it for many, many years.

Lieut.-Col. Arthur F. Hurst, M.D., R. A. M. C., London, England: I should like to point out, in reply to Dr. Prince, that my definition of hysteria differs from that of Babinski in one important particular. Babinski believes that hysteria is characterized by a pathologic state of mind which manifests itself in an abnormal liability to respond to suggestion, as a result of which symptoms are produced. In contrast to this, I believe that there is no such constant underlying mental condition and that an abnormal degree of suggestibility is merely a predisposing factor. Until actual symptoms have been produced by suggestion, hysteria is not present. I believe that failure to realize this has resulted in a very large number of cases of hysteria remaining unrecognized. My definition has the advantage of embracing all cases which are by common consent regarded as hysteria. Although some such process as dissociation, to which Dr. Prince referred, may take place in many instances, it is certainly not invariably present. Where, for example, is the dissociation in a case of hysterical vomiting, in which the vomiting is a sequel to that orginally produced by the irritation of swallowed gas? The condition is certainly hysterical, as it is produced by suggestion and is curable with extreme rapidity by psychotherapy. Such a man may have been normal in every way before he was gassed, and he is normal in every way after treatment has resulted in the cessation of vomiting. I cannot see where dissociation takes place in such a case. Dr. Prince referred to those cases of Babinski in which the history showed that such an expression of dissociation as amnesia or stupor was present at the onset. Though this was not uncommon in soldiers, a similar history was not obtainable in the majority of cases, and particularly not in those which only developed after they reached the base as the result of a suggestion which was not operative at the front.

Dr. Kennedy asked about the after-history of these patients. We always kept them in the hospital for some time after the removal of their gross hysterical symptoms, so that the effect of hard work could be observed, and any further treatment required by the presence of associated psychasthenia

or neurasthenia could be given. In the vast majority of monosymptomatic cases—such as hysterical aphonia, mutism, deafness, vomiting, and contractures—the man was sent back to the firing line after a preliminary period of home service, as it was found that apart from the particular symptom for which he was admitted to the hospital, he was perfectly fit. The subsequent histories showed that these men were not more liable to develop neuroses in the future than other soldiers. On the other hand, patients in whom a psychasthenic element was well marked and in whom there was evidence of hyperadrenalism and hyperthyroidism were rarely sent back to military duty. They were never, however, discharged from the army until they were fit to earn a living in civil life. Patients with so-called cases of shell shock, in whom hysterical symptoms developed as a sequel to an emotional experience, could be sent back to duty when they were treated at advanced casualty stations, but this could be done less often in the cases of patients admitted only after the symptoms had been allowed to become more permanently established by months of over-sympathetic treatment in hospital. Many of these men were given military duty at home, but others who had always had an unstable nervous system were discharged from the service—not, however, until they were fit to earn a living.

In reply to Dr. McCarthy: Our investigation of a very large number of cases of paralysis and contracture of the type described by Babinski as of reflex origin convinced us that at the stage in which they reached us, which was rarely less than three months after the onset and often as long as two or three years, the contracture and paralysis were invariably hysterical and removable by psychotherapy. The associated changes in the mechanical and electrical excitability of the muscles, the alterations in the deep reflexes, and the vasomotor disturbances disappeared instantaneously. The trophic changes, of course, required a much longer period before they disappeared.

12. THE MANAGEMENT OF PSYCHONEUROSIS IN THE CANADIAN ARMY. (Slides and Cinema Pictures.) Lieut.-Col. Colin K. Russel, M.D., Montreal, Que.

No discussion.

13. THE MECHANISM OF WAR NEUROSES. Dr. Sidney I. Schwab, St. Louis.

Dr. Schwab gave a definition of war neuroses. He discussed physiologic conservation as an aspect of primitive defense; the primary and secondary instincts; the emotional element; fear reactions; the rôle of polyglandular activation; neuropathic inheritance; predetermining factors; the essential trauma; the mechanism of fixation; the period of automatism, and convalescent conflict.

DISCUSSION

Dr. Hugh T. Patrick, Chicago: I think Dr. Schwab's conception of the psychoneuroses is not only correct but practical and should be carried over into civil practice. I gather that he believes that the psychoneuroses of civil life are not exactly this conservation process which constitutes the mechanism of war neuroses. In many of the civil cases, instead of using the word conservation we might use the word satisfaction, which really amounts to the same thing. The principles of treatment so successfully carried out in the army should be the goal toward which we should strive in all like conditions of civil life, though successful treatment of the latter is much more difficult.

14. THE SYMBOL AS AN ENERGY CONTAINER AND SOME SUGGESTIONS AS TO THE KINETICS OF DISCHARGE. Dr. Smith Ely Jelliffe, New York.

Dr. Jelliffe discusses the human body as an energy system. The inadequacies of the dietary theories as to energy sources are pointed out. He emphasizes the necessity for physiologists to fall back on "vital action" as an adequate explanation for the dynamics of organ action. The necessity for a search for energy sources is not usually considered as such. He discusses the development of speech itself and the kinetics of speech transformations manifested in alterations and evolutions of symbols and as substitutes for things and for conduct. The kinetics of symbolic action as discharged in thinking are also considered. They are illustrated largely by the development and evolution of the word "symbol" itself, which in its own synthesis illustrates the main features of the hypothesis assumed.

DISCUSSION

Dr. Walter Timme, New York: I pass rapidly over the physiologic and dynamic treatment of the subject with simply the statement that I believe Dr. Jelliffe does not use the word energy in the right sense. When Dr. Jelliffe talks of our receptors of "energy" and containers of "energy" he does not mean energy, but energy liberation. Energy is work done along a given path; this work within us may be in the direction of locomotion or the production of heat, or the activity of mental processes. This energy remains latent within us until it is set free—mayhap by physical stimuli, mayhap by symbolic. We produce energy within us with the help of certain agents, whose combination is simply the transmutation from extraneous supplies—such as food, air, sunshine—into easily awakened indigenous compounds. Passing over that, we then come to the various theories regarding the thymus and parathyroid in the production of tetany Uhlenhuth was compelled to advance his theory that the thymus produced a toxin because to him there was no choice left. It had been shown that an overacting thymus, when the parathyroids were normal, produced in an animal a tendency toward tetany and that tetany could be relieved by calcium salts injected into the blood vessels. If in an animal with normal thymus we remove the parathyroids, the same thing occurs, namely, a marked tendency of the animal to progress to tetany, which can again be averted by the feeding of calcium salts. In other words, the thymus produces something which conduces toward tetany in the animal, and which is excreted by means of the parathyroids. Whether or not this can be made applicable in so broad a sense as Dr. Jelliffe has presumably made use of, is left for further thought and more or less critical reading of Dr. Jelliffe's paper when published.

Dr. Smith Ely Jelliffe, New York: Just one word with reference to Dr. Timme's discussion in which I think he tends to prove the points I have tried to bring out: What we know about the transforming power of the chlorophyl system, while as yet very far from complete, does tend to point out that the kinetic vibratory light energy is transformed into a new set of combinations which we call starch, cellulose, etc. The chemical equation is fairly well known although it is not certain what part the manganese plays in the enzyme action, nor are we in precise touch with the photo-electrical processes. That the latter exist is well known. All that I have assumed is that in addition to food there are other types of stimuli or energy sources,

which in a manner analogous to the transforming capacity of the chlorophyl system, kindly offered me as an illustration by Dr. Timme, produce responses in the human being and, all taken together, have made man the organism he is, capturing, transforming and releasing these cosmic energy sources.

I then go on to try to indicate that the gradual formation of the symbol is as an energy container and how it is utilized at the intake and the outgo systems of the human beings. The visual purple of the rods and cones is an interesting analogue of the chlorophyl system, but vastly more complicated as they represent an involutionary synthesis of probably millions of years of photoreceptive experiences. This great time element during which the evolution of form as a structuralization of experience has taken place is rarely grasped by any of us, and we are all too prone to accept the impossibly trivial as adequate explanations for the enormous complexities bound up in this evolutionary mechanism. The trivial explanation is our defense reaction to prevent effort to understand these complex dynamics of life. Uhlenhuth says that feeding the larvae of certain newts up to a certain point with thymus will produce tetany-like results; we agree with him, but when he says it is due to a toxin in the thymus, we wait—the toxin hypothesis is so orthodox that we suspect its usefulness. It is so obvious that it does not explain, for what does a toxin do? How does it do it? We are no nearer an explanation of tetany when we say it is due to a thymus toxin, or that it is due, as Paton would say, to disturbance in the guanidin regulation—another type of toxemia. In these explanations the body is considered as a closed system, out of relation to other energy sources than the thymus, or guanidin or what not; whereas it is not a closed system but a closely integrated set of receptors, connectors and effectors which have been undergoing a perfecting process for many millions of years. A reaction like tetany can be understood only as a disturbance in some part or parts of this chain. Is it in the primary receptors, the peripheral arcs, the spinal arcs, the cortical arcs or the final effective muscle arcs? We have tried to show it is due to a disturbance in the calcium metabolism, or that part of it of which the parathyroid is an integral part and the particular function into which the bivalent ration enters is the electrical conductivity at the synapse at the spinal arc. The lowering of the synapse threshold prevents an orderly distribution of the cosmic stimuli, causing a flood—an avalanche. Extensor response, i. e., flight, takes place, or would take place, as is seen in the severe muscle reaction.*

15. SEGMENTAL CEREBRAL MONOPLEGIA. Dr. William G. Spiller, Philadelphia.

The author describes several cases of cerebral monoplegia in which the paralysis was confined to a small segment of a limb. This condition is likely to be confused with nerve lesion or hysteria. Such cases are not numerous in the literature.

DISCUSSION

Dr. Malcolm A. Bliss, St. Louis: This brings to my mind a case Dr. Fry had under his care in which jacksonian epilepsy of segmental type began in the feet. The sensory distress was so great that the patient would pound the feet with anything she could get hold of. The jacksonian convulsions continued up the leg in segmental progression at intervals of some weeks

*This paper will appear in full in the *Journal of Nervous and Mental Diseases.*

and eventually involved the left side of the body. There was not a great deal of positive information to be gained either from the history or from the neurologic examination. She had some complete epileptic attacks. The surgeon laid down a large parietal flap, and a tumor of the falx cerebri opposite the leg center was discovered. The condition of the patient was so serious that we stopped with the expectation of going farther at a later time. The patient developed a pyelonephritis and nothing further has been done. I thought this peculiar situation of a lesion might be of interest.

Dr. L. Pierce Clark, New York: Some years ago my attention was called to the fact that many types of epilepsy in their beginning had local paralytic equivalents, and that too, in idiopathic types rather than the organic or symptomatic jacksonian epilepsy. It might be difficult to differentiate such from Dr. Spiller's cases.

Dr. William G. Spiller, Philadelphia: Dr. Clark speaks of transitory paralysis as an epileptic equivalent. I am under the impression that Flatau was among the first to describe this, and later McConnell wrote on this subject. Transitory paralysis undoubtedly occurs following a convulsive attack in epilepsy, and it may be confined to a limb, but I cannot recall a case in which this transitory paralysis, postconvulsive or as an epileptic equivalent, was confined to a segment of a limb. If it did occur, it should cause no difficulty as regards the segmental paralysis of which I have spoken.

CHICAGO NEUROLOGICAL SOCIETY

Regular Monthly Meeting, Oct. 16, 1919

Lewis J. Pollock, M.D., *President*

A CASE OF LUMBOSACRAL PARALYSIS. Presented by Dr. G. B. Hassin.

A colored boy, aged 18, entered Cook County Hospital about two months ago, complaining of difficulty in walking. The mother stated that when he was 4 years old, he fell on the floor with the legs widely "stretched." After this he was confined to bed for nine months, but he regained the use of his legs and for ten years did not experience much difficulty in walking, though he admits that the left leg was always smaller and weaker than the right. In the last four years the left leg has steadily grown weaker and he has often fallen because of sudden flexion of the left knee. He has had measles, pneumonia, whooping cough, gonorrhea (twice) and "five sores" on the penis (about a year ago), but at the time of the accident he was perfectly well, had no fever, headache or vomiting.

Examinaton.—Marked lumbar scoliosis to the left; atrophy and shortening of the entire left lower limb; drooping of the left side of the pelvis; a slight bend of the left knee, and eversion of the left foot were present. In walking he violently bent the body forward and to the left, swung the left leg and pelvis, the toes of the left foot striking the ground. Passive movements in the joints were possible, except in the left ankle where they were somewhat limited. On the left side flexion, abduction, adduction of the foot, flexion, extension of the toes, extension of the flexed leg, adduction and flexion of the

thigh were all abolished, the only active movement possible being abduction of the thigh and flexion of the leg. The muscles implicated were all atrophied, flabby, and without fibrillary twitchings; those supplied by the obturator and posterior tibial nerves showed complete reaction of degeneration, while the muscles supplied by the anterior crural and peroneal nerve gave a normal response (only the galvanic current was available). The tendon reflexes on the left were lost, the cremasterics were very inconstant and uncertain; there were patches of anesthesia and hypesthesia scattered over the dorsum of the foot and anterolateral surface of the leg.

The roentgenographic examination revealed no bone or joint changes; the central nervous system (pupils, genito-urinary organs, mentality) was equally negative.

The clinical picture presented by this patient strongly resembled that outlined by some orthopedic surgeons (Lorenz, Bade, Peltesohn), as the result of unfortunate attempts at reduction, in small children, of congenital dislocation of the hip. Forced excessive abduction is pointed out by Bade and others as the probable cause of the lumbosacral paralysis which they think is produced by the tearing of the stretched nerves emanating from the lumbosacral plexus. The same etiologic factor was, in my opinion, the cause of the paralysis in this patient when he fell with the legs widely abducted.

FACIAL PONTILE DIPLEGIA (TRAUMATIC). Presented by Dr. G. B. Hassin.

A colored man, aged 26, had his head caught between two railroad cars. He immediately lost consciousness, which he regained, he claimed, three weeks later. About two and a half months after the injury he entered the Cook County Hospital.

Examination.—There was paralysis of both seventh nerves and of the left sixth. The face was expressionless and the lower lip hung down. The patient could not wrinkle the forehead, show the teeth, laugh, pucker the lips, close the eyes, etc. The tongue, uvula, soft palate, and the muscles that controlled chewing showed no paralysis. He complained of dryness of the tongue and mouth, had no taste for sweet, salt, bitter and sour. The seventh nerves and facial muscles showed complete reaction of degeneration. He could move the left eye in every direction except outward. Hearing was good on the left, but was somewhat impaired on the right. Douching (cold water) produced no response from the vertical canals of either ear, and there was a slow nystagmus to the left on irritation of the left horizontal canals, and past pointing to the left, 2 inches; rotation produced normal but shortened reactions from both vertical and horizontal canals (three seconds for the right and six seconds for the left ear). The roentgenographic examination revealed a fracture of the skull extending into the middle fossa, close to the base and involving the right parietotemporal region. The simultaneous involvement of the seventh and the abducens could very well be explained by a lesion of the tegmental portion of the pons where the sixth nucleus is looped by the ascending root of the facial nerve. Such a localization would also involve the longitudinal bundle and the adjacent contralateral seventh nerve which exchanges fibers with the opposite nerve. The chorda tympani changes, however, and slight hearing impairment on the right might also denote a peripheral lesion (on the right); however, this could not explain the permanent abducens paralysis on the left and the nystagmus findings.

SEX PERVERSION AND CRIME. Presented by Dr. Harry R. Hoffman.

Sex perversion is as common now as it ever has been, possibly more so. It has been estimated that there are from 75,000 to 100,000 sex perverts in Chicago. At the City House of Correction with an average of 2,000 inmates, the number of self-confessed perverts runs from fifty to 200. These persons are of such pronounced types that the attendants in the receiving room can pick them out. Nearly all of these perverts are put to work in the male laundry of the institution. The large majority are of the inverted type. Cases of exhibitionism are occasionally found, but these usually occur in debaucheés, in persons afflicted with local infirmities that explain the immodest act, or in persons afflicted with some definite mental disease, such as senile or precocious dementia. The speaker believed that many cases of prisoners sentenced for larceny and shop lifting were cases of fetishism.

The majority of the inverts in this institution show no abnormalities in physical development, but their gait, speech and tastes are feminine. They belong to the more intellectual class of prisoners. Of all the inverts now confined there, not one has failed to finish the eighth grade in school. This is remarkable considering the type of prisoners in the institution. At present there are more colored prisoners than white. These prisoners give the least trouble of any in the institution. Although the majority are recidivists, having served sentences in this or other penal institutions, they are the least troublesome. They talk freely and intimately, and they realize the depravity of their desires. They look with scorn on the other perverts, and do not care to mingle with them. The invert's love is not directed to women or children nor to other perverts, but to vigorous healthy adult males. They are intensely jealous, and it is this jealousy which often causes them to commit the crime that brings them into the penal institution.

The great majority of inverts are harmful only to themselves. Those showing criminal tendencies should early be placed in suitable institutions for mental disease. All sadists and fetishists should be handled as though they were psychoses. Those whose pervert acts are symptoms of some underlying psychosis should immediately be given institutional attention.

SOME OBSERATIONS ON DISCIPLINARY PSYCHIATRY IN THE ARMY. Presented by Dr. Herman M. Adler.

During the war, Dr. Adler had the opportunity to become familiar with the mental aspects of the problem of delinquency in the army. The army methods of dealing with delinquents are comparable to those employed in civilian life. Delinquents are confined in guardhouses pending action by court-martial, and sentences of less than six months are served in the guardhouse. Longer sentences are served in the military prisons, which are: the United States Disciplinary Barracks at Fort Leavenworth; the Atlantic Branch at Gouvernor's Island, New York, and the Pacific Branch at Alcatraz Island, San Francisco. It was estimated—on the basis of the figures for the Civil War, the Spanish-American War and the Mexican border activities—that an army of 3,000,000 men would require accommodations for 50,000 prisoners at Fort Leavenworth. Actually, the number confined in the military barracks at no time exceeded 4,000; the total number serving sentences was approximately 5,000. In time of peace, the population of the Disciplinary Barracks at Fort Leavenworth was between 1,200 and 1,700 men, corresponding to the number in the average civilian reformatory or penitentiary. Although there was an

increase of population during the war, the total number was approximately 10 per cent. of the estimated number. This result is probably, at least partially, due to: (1) The enforcement of prohibition in the cantonments of the army generally; (2) the activities of the neuropsychiatric boards at the draft examinations, and (3) the distance of the actual hostilities from the home base.

The neuropsychiatric section of the Surgeon-General's Department became effective very shortly after the declaration of hostilities, and increased in efficiency continuously during the course of the war. It was not possible, therefore, for the neuropsychiatric officers to eliminate from the army all mentally abnormal men, some of whom eventually found their way to the disciplinary barracks; nevertheless, the majority of such men were eliminated at the source. As a result, delinquency was greatly reduced, as compared with any previous experience in this country. The disciplinary problems were only a small portion of the problems considered by the neuropsychiatric section and were not, therefore, developed as rapidly as other features of the work. The unexpectedly abrupt termination of the war brought these activities to an end before the effect of the various measures determined on in the Surgeon-General's Office became fully apparent. It was possible, however, to make a special study of the mental aspects of delinquency at the Disciplinary Barracks at Fort Leavenworth. A survey of prisoners, including some 3,500 cases, was made. This included: (1) complete physical and neurologic examination; (2) mental examination; (3) psychologic rating; (4) social history, and (5) study of behavior during confinement.

The prisoners examined included all the usual types found in institutions of this sort and, in addition, about 500 men confined as conscientious objectors. The result of this survey showed that from about 10 to 12 per cent. of these men were feebleminded, and that less than 5 per cent. were insane or markedly psychopathic. This low figure, as compared with figures from civilian institutions, was undoubtedly the result of elimination by draft examinations at the cantonments.

It was found that there was a correlation between intelligence rating and education. The educational medium of the entire group fell in the seventh grammar school grade. The men in the disciplinary battalion, the organization through which the prisoners might win their way back to honorable service in the army and thereby wipe out a dishonorable discharge, on the whole rated a little higher both in intelligence and education than the general average. The religious and political conscientious objectors, so-called, were the best educated of all the prisoners and had the highest intelligence rating. The prisoners convicted of sex crimes and crimes of violence had less education and rated lower in intelligence than the other groups. The men convicted of acquisitive crimes had more education than the average and had a higher intelligence rating than any other group, except the religious and political conscientious objectors. The conscientious objectors of the alien enemy and noncitizen group were very low as to intelligence and educational rating—a third of them had no schooling and one half of them had not progressed beyond the third grade.

A personality study was conducted, dividing the prisoners into three groups: (1) Those who had been in difficulty as the result of defective intelligence or judgment, or other marked mental defects; (2) those who showed no decisive defects, but whose difficulties, either in the army or previously, resulted from a marked emotional instability, and (3) those whose difficulties could be traced to marked egocentric traits.

Sixty-six per cent. of the entire group were found to be egocentric; 24 per cent. inadequate, and 6 per cent. emotionally unstable. The small number of the last group was explained on the assumption that delinquents of this type usually arouse sympathy in their fellowmen and are rarely sentenced to confinement. The inadequate group included not only the feebleminded, but the higher grades of defectives. The egocentric type expressed itself by insubordination, arrogance, an uncompromising attitude, vanity, lack of emotional generosity, and, above all, selfishness. The emotonal make-up that went with this resulted in violent or unstable behavior and was dominated by one or more of the factors mentioned above. It would appear from these studies that this type of personality was the one that most frequently caused trouble in army life, and that it was at the bottom of about three fourths of the disciplinary difficulties and delinquencies. So far as the treatment was concerned, the survey indicated that the experiences with similar personality disturbances observed in the hospitals for mental diseases were applicable. The inadequate group required suggestion from a stronger and more intelligent personality. The emotionally unstable group required care during their emotional upsets, and reassurance and discipline of habit at other times. The egocentric group were best managed by an impersonal and logical attitude on the part of the authorities. Any attempt to change the type of personality, either by logical argument, or by coercion with force or otherwise, led to an increase in the egocentric manifestations. The sane method of persuasion and of impersonal attitude toward the inviolability of individuality in the end secured the prisoners' concurrence, if not their cooperation.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

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GEORGE A. WATERMAN, M.D., *President*

THERAPEUTIC PROCEDURES FOR THE RELIEF OF DECREASED INHIBITION.

Presented by DR. JOHN BRYANT.

In animals, and in man, life is difficult or almost impossible without reasonably normal development and action of the central inhibitory mechanism. The world is full of patients, mostly chronic, in whom inhibition is decreased. One obvious, ultimate result of this decreased inhibition is chronic exhaustion. In order to relieve chronic exhaustion, its cause must be attacked. In diet and exercise, properly utilized, we have two valuable and apparently too little appreciated methods of attack on the problem of decreased inhibition. The first essential in the matter of diet is the temporarily complete elimination of eggs, meat and fish, and the restriction of milk to a few ounces daily. This strict diet is maintained for from two weeks to a month. At the close of this period, there is a gradual resumption of general diet, but on an intermittent basis. The second week there may be two days of unrestricted diet, and in the third progressive week the patient may arrive at unrestricted diet every other day. It is unlikely, however, that such a patient can ever profitably take unrestricted diet on continuous days for any great length of time without suffering harm from excessive chemical stimulation. The employment of physical exercise has for its immediate object the sharpening of muscle sense perception. Such exercise is carried out at the very slow rate of about ten

seconds per linear foot of distance. One simple exercise for each main group has been developed, and in the army a routine was employed which took for its full completion about one hour of time. Parallel with the increasing power of physical control comes a noticeable increase in mental poise and the evaporation of mild phobias which have as their basis the inabilty to handle properly the locomotor mechanism. The two most usual phobias observed have been the fear of high places and the fear of crossing streets when there is city traffic.

DISCUSSION

DR. MORTON PRINCE: I wonder if Dr. Bryant would state more clearly and in detail the class of cases to which he refers.

DR. JOHN BRYANT: The patients that I have been working with are mostly the asthenics and the enteroptotics. They are almost always in a state of chronic exhaustion. They come to me because they have some intestinal trouble, but as I work with them I find that I must consider the general physical development and eliminate some of the mental abnormalities before I can produce results. I saw this afternoon a working man who, when he came to me, could not get through his day's work. Now, after two months, he is hard at work and goes home feeling fresh instead of exhausted, and he sleeps soundly instead of staying up all night complaining of his gastro-intestinal tract.

DR. E. W. TAYLOR: One of the chief values of this communication, it seems to me, is what we have been seeing recently in so many branches of medicine—closer relationship between the neurologic side of disease and certain definite physical phenomena which ordinarily have been included in the general category of internal medicine. On every hand, we are observing this integration, as it were, in these branches of medicine, particularly in relation to the nervous system. No doubt the war has had much to do with bringing this out.

TRANSMISSION OF BREATHING THROUGH THE HEAD. Presented by DR. A. MYERSON.

In various parts of the head, and especially in persons without much hair, breathing sounds can be easily heard with a stethoscope applied in the usual manner. The points at which they can be heard best are: First, over the antrum, where they are loud; second, over the temples where they are less clear in some individuals, but where in many cases they are quite distinct; third, on the vertex, where in bald-headed men they are as distinct as over the temples; fourth, on each side of the occiput; and fifth, just below the occiput in the upper cervical region, near the spinal column where they are very distinct. The sounds heard are blowing in character, with prolonged expiration. It is sometimes necessary to ask the subject to breathe deeply. The sounds are unquestionably transmitted directly through the skull, and very likely come from the upper nasal, pharyngeal and laryngeal parts of the respiratory tract. This is reported as a purely physiologic phenomenon constantly present whenever conditions make it possible to get close to the skull. No pathologic relationship has as yet been investigated.

NEUROLOGIC AND PSYCHIATRIC WORK AT SAVENAY. Presented by DR. H. M. SWIFT.

The hospital center of the American Expeditionary Forces at Savenay comprised a number of base hospital units, one of which was Base Hospital No. 214,

used exclusively for the care and evacuation of patients whose cases were previously diagnosed as nervous or mental. The types most frequently seen were those of mental deficiency, various psychoses, psychoneuroses, epilepsy and constitutional psychopathic states. There were also admitted many patients with organic nervous conditions, of which perhaps the most noteworthy were the residuals of encephalitis lethargica.

The mental defectives might be roughly divided into the physically strong and the physically weak. Previous to the armistice, mental defectives of good physical condition and without conduct abnormality were usually reclassified and recommended for labor organizations. Frequently, mental deficiency was found to be associated with congenital physical deficiency manifesting itself by generally poor bodily structure, often with narrow chest, stooping shoulders and flabby abdominal wall (the type designated by some as the carnivorous type). For these persons with associated congenital mental and physical deficiency the term constitutional deficiency would seem more descriptive than mental deficiency and would not imply so great a stigma. In this group, evidence of a generally poor nervous system was shown by the small resistance to stress and fatigue with resulting neurasthenic states.

The psychoses did not differ strikingly from the psychoses of civil life. Interesting were the cases of soldiers with clinical pictures resembling that of dementia praecox who subsequently made apparent recoveries. Of manic depressive insanity, maniacal states were relatively more common than among a corresponding male population in civil hospitals for the insane.

The psychoneuroses comprised the war neuroses, or those arising in connection with shell explosion, and neuroses occurring independent of so definite a causal factor. The latter did not differ essentially from the neuroses of civil life. In both groups, the constitutional element was frequently evident.

Epilepsy: Of especial interest were cases of epilepsy developing in the service with a history of no attacks previous to enlistment. It is suggested that in addition to the ordinary type of epilepsy manifesting itself in the form of convulsions, etc., there may also exist persons with a latent epileptic tendency which under ordinary conditions may never come to the surface, but which becomes active under especially unfavorable influences, such as may be incident to army life.

The term constitutional psychopathic state was rather loosely employed to designate various character abnormalities. In this term were included the cases of many men who by reason of native peculiarity or defect showed themselves inadaptable to army life. Alcoholics and many delinquents were also classified under this heading.

Encephalitis Lethargica: At Base Hospital No. 214 only residuals were seen, chiefly cases of cranial nerve palsies and other cases of a peculiar condition resembling paralysis agitans with stiffness and with mask-like facies. The cranial nerve palsies were undoubtedly nuclear. The oculomotor and the facial nerves were most frequently affected. The frequency of nuclear lesions suggests that the inflammatory process has a tendency to damage more particularly gray matter. The state that resembled paralysis agitans might be explained perhaps on the basis of damage to some hypothetical nucleus that normally has a regulatory influence on the motor system, the lesion leaving as a resultant the peculiar symptoms noted.

Several cases of flaccid paralysis of the legs following infectious disease were seen which were diagnosed as peripheral neuritis, but which might have been due to an anterior horn lesion. They differed from the ordinary periph-

eral neuritis of so severe a grade in the rapidity of improvement; in this respect they resembled some of the cranial nerve palsies.

May it not be that the inflammatory process of encephalitis lethargica is really of the nature of a polioencephalomyelitis and that the symptoms vary according to the region of the central nervous system most seriously affected? In many of the cases a history of a lethargic state could be obtained. This seemed sometimes to have followed influenza. In other cases the history was indefinite. In one case of the paralysis agitans type, although the stiffness seemed to have been of sudden onset, the patient maintained that otherwise he had not been ill. Yet the clinical picture was almost precisely the same as that in other cases with a definite history of an acute febrile state.

NEUROPSYCHIATRIC EXPERIENCES AT VICHY AND SAVENAY.

Presented by DR. DONALD J. MACPHERSON.

During the months of October, November and December, 1918, 124 brains and 119 spinal cords were examined and preserved at the Neuropathological Department of the Central Laboratory at Vichy. In twenty-two cases gross lesions were observed. There were eleven cases of meningitis, of which seven were secondary to gunshot wounds of the head or spinal column. In three cases, injury of the lumbar or sacral portion of the spinal canal was followed by a diffuse meningitis involving the cord, and base and vertex of the brain. Five cases of pneumonia were complicated by meningeal hemorrhage. In one case presenting the clinical picture of a transverse myelitis with paraplegia, following penetration of the back one-half inch to the left of the vertebral column by a small piece of shrapnel, the vertebrae were found uninjured and the dura intact. One and a half inches of the cord in this region appeared as a soft, flattened mass. There was indirect traumatic softening. Other pathologic findings included cases with tetanus, syringomyelia, pituitary tumor and one with a small cholesteatoma.

An analysis of 1,760 cases classified as mental or neurologic at the Savenay "triage" to March 1, 1919, showed 1,436 injuries to nerves of the extremities. Of these, 609 were on the left side of the body, 544 on the right side, and 283 were not classified as to location. Graphic charts showed the relative greater frequency of injuries to nerves of the upper extremities. There were eighty cases with injuries involving the central nervous system, twenty-nine injuries to cranial nerves, and fifty-six miscellaneous affections including syphilis, epilepsy, multiple sclerosis, brain tumor, neuritis, etc. Only twenty-nine functional cases and one psychosis were reported. One hundred and twenty cases were unclassified. These were largely cases in which complete examination was difficult because of splints.

DR. JOHN BRYANT: I was asked whether I would describe the kind of patient that I was dealing with in this paper. I think the type has been well described by Dr. Swift. It is the same type of patient as that of which he speaks—the congenitally deficient patient—only he is referring to those in whom the mental element is more pronounced than the physical element. The similarity of the two types of cases is evidenced by the fact that Dr. Swift described them by the word "carnivorous," a term for which I am primarily responsible.

DR. GEORGE A. WATERMAN: This question of discipline was one of the most difficult problems with which we had to deal at Parker Hill. We were often

asked to see a man reported because, for example, he had given the sergeant a knock-out blow. Those things were fairly simply dealt with when they occurred near the front and when the army was in operation, but when the soldiers reached us they were simply waiting to be dismissed and discipline had become very lax. They were more or less filled with the idea that they were heroes, and the attitude of the commanding officer was that strict discipline was sure to bring newspaper notoriety. The men wanted to be dismissed and it was very difficult to send them to hospitals for neuroses. We had men from the tenderloin, for instance, who had never had any discipline and who were distinctly of a psychopathic constitution; such men were very difficult to manage.

Dr. J. W. Courtney: I should like to ask Dr. Swift, first, whether in his rather large group of cases of encephalitis lethargica there were fundus examinations and if so, what those examinations revealed; and, second, as to what the exact syndrome was in those cases which were afterward recorded as "psychoses, unclassified," but which were referred to him under what I consider the most ill-chosen and unfortunate of all psychiatric designations—dementia praecox.

Dr. H. M. Swift: In the encephalitis lethargic cases, the fundus when examined was found normal. The symptoms in the cases resembling dementia praecox varied greatly and many times were fairly well cleared up when the patients reached us. We sometimes had a history of a catatonic condition. Other patients were more of a paranoid type, that is, they had delusions, not very well systematized, which very frequently improved. For that reason, we were always reluctant about making the diagnosis of dementia praecox because that rather implied the disease had existed previous to enlistment and we thought that those patients in whom the condition had developed while they were in the service were entitled to compensation as for an injury received in line of duty.

Dr. Morton Prince: I should like to ask a question; but before I ask it I should like, apropos of dementia praecox, to mention a case. I think it was one of the worst cases I have ever seen. The patient was sent out to Waverley, and after being studied a long time the diagnosis of dementia praecox was made. It was a terrible case, so bad that the family was told that one could only hope that complete dementia would come soon and so end the mental distress. Recently, while overseas, I had a letter from the young lady saying that she is perfectly well. (Dr. Waterman, who also was concerned in the case, confirms this report.) Hence a patient with a most severe case of dementia praecox may recover.

The question I was going to ask is this: At the last meeting of this Society that I attended before going abroad, the subject of examinations to weed out the feebleminded from the drafted men was discussed. I recall that the members of the Society were explaining with some enthusiasm the results of their methods for the detection of cases of mental inferiority, etc. I also remember that Dr. Fernald said that about twenty-seven of his patients had been accepted by the army. Dr. Knapp then asked "if he had hung out a service flag from his institution?" How was it these morons of whom Dr. Swift speaks got into the army? Were the bars let down or were they not detected in spite of the mental tests when they passed their examinations in various parts of the country? How was it they got in, and when they were in did they do good work? You said some were sent to the army labor

organizations. The psychiatrists on the examining boards were reporting their wonderful results, and the neurologists were reporting their results, and here we have these large numbers of defectives in the army.

Dr. W. H. Prescott: In this connection I may say that we were asked to see a patient at the psychopathic hospital whom we found to be a man that had escaped from the Worcester Hospital three months before, and that had been admitted to the army within a month from the time he had escaped. He was a most marked example of dementia, and did not seem to be able to tell where he had been or what he was doing. He was immediately sent back to Worcester. How he got into the army I have never been able to understand because his face showed what he was.

Dr. A. E. Brownrigg: Apparently all these cases of mental deficiency and other abnormalities were not detected on the first examination by army physicians, and yet I think those who have seen a great many cases of mental deficiency in the army cannot help looking with admiration at the results that have been achieved by the neuropsychiatric units. I was stationed at Fort Porter. Here were received practically all the patients suffering from mental disturbances that came to this country, and I presume that individually I saw more of these returned patients than any man in this country. In considering these men, a large number, as Dr. Swift has intimated, were found to be mental defectives. I have no statistics, but I have a clear memory that the majority of these patients came from the South, Louisiana and Mississippi, and also a large group came from Minnesota and Wisconsin. I gathered from correspondence in trying to place them back in their homes and from their service records that most of these patients came into the army before the neuropsychiatric units were operating. We investigated these cases time and time again and found that these men were sent overseas in the early months of the war. We know that Dr. Pearce Bailey and the members of his department were not actively concerned in examining these units until late in 1917 or early in 1918. I think, therefore, that of the troops that came through in 1918 that received the neuropsychiatric examination, we did not receive any of the low grade imbeciles, and it was only in those early enlistments that we occasionally saw them. I remember one patient in particular. I sent him home with an attendant because he did not know enough to travel alone. He said papa had always driven him to town. He graded six years and had never been allowed away from home. He was 37 years old, and anybody would know that he was a born fool. He was not, of course, a good soldier, and he spent his time in hospitals.

The psychopathic patients were called "incorrigibles;" they were not adaptable to army life and it was very hard to have them discharged honorably.

The patients with dementia praecox had all the cardinal symptoms of dementia praecox under the old classification of Kraepelin. They were sometimes brought to us on stretchers and with special guards. Some were paranoid and some were of the simple dementia form. But the remarkable fact was that after a stay of three or four months, a large proportion of them became well enough to be returned home, sometimes without an attendant, and they made a very good social recovery. Many years ago I read a paper before this Society in which I suggested the improvement and cure of dementia praecox. I never thought that the dementia in dementia praecox meant that a certain patient was always absolutely demented, but that he had dementia

praecox in much the same real way that one might be without severe typhoid symptoms, but nevertheless have a true attack. I think that this experience in the army made me believe that whether considered to be a psychogenic disease wholly or one that is on a defective basis, the patients that lose control of their minds are curable and recover if treated early. I think this experience in the army has proved the truth of such a general statement.

DR. H. M. SWIFT: In answer to Dr. Prince, I should like to say that although we had a great number of mental cases, both defective and psychotic, yet relative to the total number of men who went over I think the number was probably rather small.